This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

Reg. U. S. Pat. Off.

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Industrial Dermatoses

—*Due to Fungi*—

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TO DECIDE the occupational origin of fungous diseases, the following must be considered:

1. Was the infection contracted outside of working hours?
2. Did conditions exist in the industry which could have caused infection of the unbroken skin?
3. Did an injury or trauma in the individual's particular occupation lay the skin open to fungous infection?

Ringworm, commonly known as "athlete's foot," can be attributed to occupation only when it directly attacks such employees as attendants who clean swimming pools in bare feet.

The most serious and baffling problem is raised by the dermatophytids of the hands that occur in connection with dermatophytosis on distant parts.

A dermatophytid from foot ringworm manifests itself as an eczema of the hands which is scaling, vesicular, or bullous. It so often resembles a contact dermatitis that differentiation between the two is difficult. However, the following elaborate criteria to verify the diagnosis would be impractical in every case:

1. The presence of erythematous soggy and macerated skin in the toe webs.
2. Scrapings from the lesion positive for fungi microscopically or in culture.
3. A positive trichophytin test with reservations as to its interpretation since its diagnostic value is doubtful and is group specific only. It merely implies the presence of a little understood form of immunity.
4. Ability to reproduce in controls a similar allergic eruption of the hands by implanation of fungi in the toe webs.
5. Recovery of fungi in culture from the blood of same species as found on the feet.

I believe that the time element is the important factor in this problem. A contact dermatitis should clear up within a few days or a few weeks after the irritant is removed.

It is possible for the physician who sees the case some months after the onset of the eruption with the history that the dermatitis of the hands has remained stationary, that it is localized and grouped instead of diffuse, that there are scales and soggy skin in the toe webs containing fungi, and that recessions and exacerbations occur in the same locations, to conclude that it is not an occupational disease.

Ringworm is one of the most widespread of the fungous diseases of human beings. It is produced by the various members of the Trichophyton, Microsporon and Epidermophyton groups. Trichophytons attack chiefly the hair, Epidermophytons the glabrous skin, and Microsporons both the hairy and glabrous areas.

SINCE this subject is such a vast one, I will give the following classification, to simplify matters, since they are the varieties with which we are mainly concerned:

TRICHOPHYTONS

T. Plicatile.
  a. violaceum, b. crateriforme,
  c. accuminatum.
T. Neo-endothrix.
T. Ectothrix:
  a. small spore variety: T. gypseum (T. gypseum from horses and T. gypseum from cattle frequently affect the beard in man).
  b. large spore variety: T. niveum,
     T. rosaceum,
     T. faviforme.
T. Cerebriforme.
T. Picatile.

MICROSPORONS

M. Adouini, of human origin.
M. Lanosum (Caninum), of animal origin.

EPIDERMOPHYTONS

E. Interdigitale—commonest cause of athlete's foot.
E. Inguinale, isolated in Tinea Cruris.

DERMATOPHYTOSIS (ringworm) of the hands and feet. In its mild form it may show some scaling and superficial fissuring in the toe webs, especially in the fourth interspace. Tense vesicles, bullae, and occasionally pustules may occur on the sides of the feet and the soles. Heaping up of crusts, eczema and keratoses are other types of lesions seen.

EPIDERMOPHYTIDS (allergic manifestation) commonly occur on the hands in the form of non-inflammatory minute clear vesicles. They may become grouped, dry and crusted, and heal spontaneously only to be succeeded by new crops which go through a similar cycle. Such localized patches and arrangement differentiate this from a contact dermatitis which is invariably diffuse. It is the epidermophytids which are often mistaken by workers for a contact dermatitis of occupational origin and give rise to diagnostic dispute on the part of physicians. An eruption which recurs weeks or months after the patient has stopped working is, in my opinion, not due to occupational causes.

Another concept advanced by some in this connection is that the presence of an epidermophytid produces polyvalent sensitivity so that the patient reacts to chemical substances to which under ordinary circumstances he would have been immune.

This is unfounded in fact, and no satisfactory evidence for this claim has been offered.

In fact, I believe that the only influence of epidermophytids is merely that of a break in the skin which may permit irritants to produce a dermatitis.

TINEA CIRCINATA (Trichophytosis Corporis, Herpes Tonsurans or Ringworm of the Body). The dry or macular type begins as a small red spot slightly raised and spreads peripherally to form a ring. The lesion consists of a border with transitory vesicles and a clearing, sightly scaly centre. It may be accompanied by slight itching or burning.

In the moist or vesiculo-pustular type the vesicles may arise irregularly or in a ring just behind which the margin is red and edematous. These dry rapidly and form crusts followed by spontaneous healing. Infections of the hair follicles produce pustules. A Trichophyton or Microsporon, usually lanosum, is the organism most frequently isolated.

TINEA CRURIS (eczema marginatum). This occurs on the inner sides of the thighs and the perineum. Heat, perspiration and friction are predisposing factors, and hence is not uncommon among cavalrymen and others who spend much time on horseback. It is caused principally by the Epidermophyton Inguinale and is characterized by bright red patches with elevated scaly borders distinctly marked off from the surrounding skin. Dhobie itch is the name given to it in India where it occurs among washerwomen.

Yeast infections are similar to those of ringworm and occur in the interdigital webs, groins, internatal cleft, submammary folds, axillae and nail folds.

BLASTOMYCETES (yeast-like fungi) which are pathogenic both to animals and man, are responsible for the great majority of industrial fungous affections. Castellani has classified these organisms morphologically as follows:

Cryptococcus, having no mycelium and no asci.
Saccharomyces, having asci but no mycelium.
Monilia, having mycelium but no asci.
Endomyces, having both mycelium and asci.

BLASTOMYCOSIS (Blastomycetic dermatitis, Saccharomycosis hominis or Dermatitis Blastomycetica). Cutaneous blastomycosis is a chronic inflammatory disease beginning as a small papule or papulo-pustule, then becoming crusted and slowly extending peripherally, forming a well defined elevated verrucous patch on a base infiltrated with pus. The lesions have a characteristic dark red sharply sloping border filled with minute deep-seated abscesses. After several months duration the larger lesions may be partly covered by thick adherent crusts beneath which are large lobulated papillary projections suffused with sero-purulent exudate. The lesions extend peripherally and tend to heal in the centre with thick scar-like formation.

ETIOLOGY: The causative organism is classified as a cryptococcus (gilchrist) or an oidium (dermatitidis) and is a yeast-like fungus. The term blastomyces is a very vague and confused one. Infection occurs through direct contact with the skin. The exposed parts are usually involved although no part of the skin is exempt. In systemic blastomycosis any of the organs or bones may be involved. Multiple subcutaneous nodules and abscesses associated with temperature and constitutional symptoms occur. The prognosis is grave, a mortality of 90% having been thus far reported.

EPIZOOTIC LYMPHANGITIS is due to a cryptococcic farcinous blastomycete transmitted from cattle to man.

CHROMOBLASTOMYCOSIS is an infection similar to blastomycosis histologically and is due to a fungous first recognized by Lane in 1915. It is the Phialophora verrucosa. This fungous does not grow by budding in tissue and has a dark brown color in culture. It manifests itself as violasceous nodules or large vegetative tumors resembling tuberculosis verrucosa cutis. It is supposed to be transmitted through injury by vegetable fragments.

COCCIDOIDAL GRANULOMA is a disease also resembling the systemic form of blastomycosis. It is caused by Coccidoides immitis and appears on the skin in primary lesions as papules, nodules, pustules, and vegetating papillomatous lesions. Ulcers and abscesses discharging a mucoid greyish-yellow pus containing the fungous appear subsequently. Most of the cases reported in this country have occurred in the San Joaquin Valley, California. It has also been described as "Valley Fever" by Dickson.

SPOROTRICHOSIS: Clinically this disease is characterized by the formation of indolent cutaneous nodules, abscesses and painless ulcers arranged in a line or chain along a lymph vessel with associated involvement of the mucous membrane, muscles and bones. The *sporotrichum* is the causative

organism and is frequently transmitted to man by the handling of the barberry bush (Berberis thunbergii).

NOCARDIOSIS CUTIS is identical with sporotrichosis but is due to an actinomyces.

ACTINOMYCOSIS (Lumpy Jaw) is caused by the ray fungus which is parasitic on many kinds of plants and pathogenic to animals and man. Most of the reported cases arise in open wounds or sores from contact with the infected grain although the disease may also be transmitted in rare cases directly from a diseased animal or through its milk or raw meat. Inoculation is frequently conveyed through a decayed tooth or through the respiratory or alimentary tracts. The majority of the cases in human beings are of occupational origin, and occur chiefly among farmers, laborers, coachmen, grooms, and millers.

EROSIO INTERDIGITALIS BLASTOMYCETIDA (Intertrigo Saccharomycetica) is a mycotic infection occurring usually in the web between the middle and third fingers among persons who are in frequent contact with dishwater. The skin becomes soggy, white, thickened and eroded with some erythema around the borders of the lesions.

MYCOTIC PARONYCHIA is characterized by suppuration and erythema around the nail matrix with resulting dystrophy. The nail fold swells, tiny abscesses may form under surface of the nail just inside the margin from which drops of pus can be expressed. This is frequently seen among women who keep their hands in water or syrups a good deal. It is also seen among bartenders, countermen, soda clerks, etc.

Occupational Sources of Fungous Infections

ANIMALS: Domestic animals and poultry may infect man with various fungous diseases, often of a particularly virulent type. The *Trichophyton ectothrix* is communicable to man from horses, cattle, sheep, pigs, deer, dogs and birds, and also from hides and horse-blankets. This organism causes ringworm of the beard, nails, and body, but rarely attacks the scalp. When the disease is caught from horses and cattle the lesions are usually raised well defined discs or nodules covered with pustules. Such infections heal spontaneously, and the first attack appears to provide immunity thereafter. The infection can also be transmitted from one human being to another, e.g., barbers may contract it from their customers. Workmen on stock farms, coachmen, stablemen, dairymen and employees in the hide and leather industry are subject to direct infection.

The small spored *ectothrix trichophyton* occurs in dogs and horses, and the large spored variety in cattle, among whom the faviform type is common. From diseased sheep skins, shearers and sheep tenders may contract an acute suppurative sycosis of the chin and neck.

Cats and sometimes dogs are the source of ringworm infections. *Microsporon* is common among cats and when transferred to man produces well marked inflammatory papular or vesicular patches. In one British Government Department, 194 women were affected by an epidemic of ringworm believed to be of cat origin. *Microsporon felineum, equinum,* and *lanosum* attack only the glabrous skin and beard in adults. The only *Neo-endothrix* communicable to man from animals is *T. cerebriforme.* Numerous cases have been reported in which ringworm has been spread by cats and dogs in private families, and domestic employees may thus incur the infection as an industrial disease. Workers in pet shops, kennels, homes for stray animals, and veterinary hospitals are similarly subject to ringworm.

Fowl, canaries, and pigeons can carry large-spored fungus, *T. rosaceum,* which attacks the scalp and beard of man, producing vesico-pustules, suppuration, and kerion. Cultures of this organism are rose-pink in color. R. Prosser White cites the case of a woman who contracted favus from a pet canary which exhibited typical cup-shaped lesions on its neck and head. Pet shop keepers, pigeon fanciers, workers on poultry farms, and persons who dress, transport and sell poultry are exposed to such infections.

Mice are frequent carriers of fungous infections, particularly favus. The organism is most often *Achorion quinckeanum.* In Australia plagues of mice have been coincident with epidemics of fungous infections among human beings. An instance is reported from Amsterdam in which girls employed in a clothing store developed a very persistent eruption on the hands from which *Achorion quinckeanum* was isolated. Mice caught in the establishment showed whitish spots on the head, and cultures from these yielded the same fungus. Numerous cases have been published reporting favus contracted from mice by infants and children.

Blastomycosis frequently occurs among persons whose work brings them in contact with animals. Ravogli has reported cases of this disease in two stable boys, two sheep breeders, a laborer in a slaughter house, and a colored woman cowherd.

Sporotrichosis has been observed in the horse, the mule, and the dog, and a case is on record in which a boy contracted it from tending a cow.

PLANTS: Yeast-like organisms are most commonly transmitted to man by vegetable matter. Straw cutters, paper makers, farmers, horiculturists and wood choppers are subject to blastomycosis through contact with the materials of their work.

Peasants working in the beet fields in the neighborhood of Prague were frequently affected with blastomycosis of the hands and feet, and true *Blastomycetes* were cultivated from the surface of the growing beet-plant. A man handling grain in a grain elevator contracted blastomycosis of the lip which was mistaken for cancer.

SPOROTRICHUM grows on many plants from which it infects the hands of persons cultivating or otherwise handling them, usually through a break in the skin. Farmers, horticulturists, and grain or malt handlers are subject to the infection, and less frequently florists, schoolteachers, berry pickers, and potato handlers. A number of cases of sporotrich-

osis have been reported among gardeners, 10 instances having occurred in one Wisconsin nursery from handling the barberry shrub. A housewife is reported to have developed the disease from picking straw.

ACTINOMYCETES grow on herbs and grain, and can infect any open sore. They are unable to remain virulent for more than one year on vegetable matter. A woman laborer who cut her hand while peeling potatoes developed actinomycosis which remained localized to the site of the wound. A more serious affection was reported in a woman who worked in a wheat field and had charge of cattle. While tying sheaves of wheat her skin was pierced by a spike of the plant and a painful edematous lesion developed around the wound. This was followed by vesicles and abscess formation, and later by deep sinuses and crateriform ulcers. The condition culminated in periostitis and bone necrosis.

FRUITS: Workers who handle fruit in the canning industry are subject to monilia infections resulting in interdigital erosions and chronic paronychia. Picking, peeling, and coring fruits such as pears, apples, and strawberries give rise to a mycotic eruption from a yeast fungus whose exact classification is not known. Kingery and Thienes report that one-third of the employees in canning factories contract a suppurative paronychia during the pear season with frequent loss of the entire finger-nails.

Sutherland-Campbell in 1929 demonstrated a variety of onychia among workers engaged in pressing oranges by hand for commercial orange juice. The oranges which caused the infection were of an inferior grade known to the trade as "culls," and were shrunken and discolored by the action of the infective organism. The lesions attacked the nails of both hands, which first showed punctiform erosions and later became yellow and friable. The surrounding skin was red and swollen. The lesions were painless except on pressure, which in some instances yielded pus from the ungual groove. Sutherland-Campbell attributed the condition to a parasite which he considered a spore form of a toadstool belonging to the Mucoracae. The periungual lesions were similar to those reported by Kingery and Thienes, who isolated yeasts of the Monilia type in their cases.

Onychia among workers in the manufacture of crystallized fruits, who are subject to constant maceration of the hands in hot and cold water, fruit juices, syrups, and sugar, has in some cases been attributed to a yeast fungus.

MOLDS capable of causing a rash on the forehead, face, dorsa of the hands, and scrotum are frequently conveyed to the skin of workers among plant and vegetable materials.

Sorters and packers of dried fruits have been subject to cutaneous irritation from molds. Several cases have been reported among women in the Orient, who developed an itching infection of the wrists by rubbing against the sacking on the tables containing the fruits. The mold isolated was the *Cereosporella vexans,* resembling *Trichophyton asteroides.*

A black, powdery mold coats the macerated dry stalks of sugar cane and produces eruptions of the skin among some of the workmen who handle it. A white mold affects basket makers who split, hammer, and cut the calamus rotang, or calamus draco (The Elanewel of the West Indies), which is supposed to contain the substance called "dragon's blood." Numerous painful fissures develop on the skin, chiefly in the palms and fingers, with cracks running longitudinally but never in the transverse creases.

A white powder from the canes or rhizomes of large reeds of Provence (Arundo donax), which are used in the basket trade and also as laths in the ceiling of houses, can cause cutaneous eruptions among workers who strip the bark from them. The irritation is generally accompanied by fever, headache, slight lumbago, and sometimes conjunctivitis, rhinitis, pharyngitis, and even acute bronchitis. The cutaneous lesions begin with erythematous plaques and slight swelling. In a few days groups of fine more or less confluent vesicles appear which are later replaced by crusts. Rarely, they become pustular. The pruritus is intense and there is a sensation of smarting. Serra has demonstrated that the eruption is due to spores of a toadstool, which develop in the canes when they are piled up in humid places. He has classed this toadstool in the family of *Ustilaginae,* under the name of *Ustilago hypodites.* Some authors consider it to be *Sporotrichum dermatodes.* This occupational risk can easily be avoided by sprinkling the canes with water to prevent dissemination of the mycotic dust.

A similar eruption often occurs among workers who bind or vend hay, straw, or rushes and among persons who make cane-bottomed chairs or wrap bottles in matting. This is thought to be due to the *Scopulariopsis koninjii.* which occurs in litter, manure, and straw.

Raw cotton is frequently contaminated by dust containing molds. The cotton tester must put his hands far down into the baled raw cotton to remove a sample, and the dust arising from it may create a transitory rash. Cotton sorters are also exposed to this affection.

Mycosis from *Aspergillus niger* or flavus has been said to occur in the external meatus of the ear among weavers, spinners, wool sorters, paper mill workers, and laundry workers.

MISCELLANEOUS SOURCES: Fungi grow on silk, wool, and leather. Ravogli reported on the case of a cobbler who contracted blastomycosis from repairing shoes infected with the organism.

Scopulariopsis brevicaulis is a fungus found in decaying and decomposing vegetation, paper, and leather. It may infect the nails or produce a vesicular eruption of the skin, nodules, granulomata, or ulcers as recently described by Markley, Philpott and Weidman.

Wooden floors and fixtures, brushes, combs, bath-

tubs, towels and clothing readily transmit ringworm and other mycotic infections.

Continuous immersion of the skin in contaminated water may give rise to mycoses. The lesions usually show grouped herpetic vesicles surrounded by a red ring and possibly pustules. Rupture of the vesicles discloses a red weeping surface which is followed by scaling. Sores on the hands of charwomen, bartenders, waiters and dishwashers frequently become infected with fungi.

Schwartz reported 12 cases of paronychia among housewives, domestic servants, and dishwashers. Monilia was found, although he did not claim that this organism was the causative agent. Roetter mentioned 12 washerwomen affected with continual maceration and irritation by strong alkalis and fats contained in the soaps.

Dodge cites the occurrence of interdigital blastomycosis among paper workers in the province of Gifu.

In the manufacture of *mino* paper, the workers keep their hands in water practically the whole day. The paper made from the bark of *Broussonentia papyrifera Vent.* or *Quercus japonicus Miq.* or *H. manihot L.* is added to the slime which holds the paper fibres together to make it suitable for use in umbrellas. Of 50 workers examined, 30 had interdigital blastomycosis of the hands. The same fungus has been isolated from the raw root of Hibiscus, from the preparation used in paper making, and from the lesions of the paper makers.

Complete destruction of the finger-nails has been reported among employees in breweries who scrape masses of yeast from fermenting casks with their finger-nails.

Crusted growths form under the nails, and produce numerous lacunae.

WOOD has transmitted various fungi to man. A case of sporotrichosis occurred in a carpenter who mended the manger of an infected horse. R. Prosser White reported cases of sudden acute erythematous rash on the face, hands and arms in two porters in an infirmary, who swept a cellar containing a large amount of dry decaying wood. The lesions were believed to be due to the dry-rot fungous.

Urticarial itch among dockworkers handling logs of African mahogany (Rungus) wood was reported by Mense. The eruption was produced by hyphomycetes which infested the bark, and no longer occurred after these fungi were destroyed by steam.

Blastomycosis has been reported among wood choppers engaged in making packing cases in this country.

References:

1. DE ALMEIDA, FLORIANO: (Univ. of Sao Paulo), Chromomycosis, *Arch. de biol.* 20:68-71, June, 1936.

2. BLAIR, JACKSON, and YARIAN, NORMAN C.: Two cases of Sporotricosis Infection due to Barberry, *J.A.M.A.* 91:96 (July 14) 1928.

3. BRUMPT, E.: *Precis de parisitologie.* Partis, Masson et Cie., 1922, p. 1168.

4. CARRION, A. L.: Chromoblastomycosis, *Puerto Rico J. of P.H. and Trop. Med.* 10:543 (June) 1936.

5. DICKSON, ERNEST C.: *Calif. & West. Med.* 47:151-155 (September) 1937.

6. DODGE, C. W.: *Medical Mycology.* St. Louis, Mo., C. V. Mosby Co., 1935, p. 288.

7. DOWNING, J. G.: Industrial Dermatoses, *J. Indust. Hyg.* 17:138 (July) 1935.

8. EAMMONS, C. W., and CARRION, A. L.: The Phialophora Type of Sporulation in *Hormodendrum pedrosoi* and *Hormodendrum Compactum, Puerto Rico J. of P.H. and Trop. Med.* 11:703-710 (June) 1936.

9. FARNBURG, S. J. and KAUFMAN, J. G.: *J.A.M.A.* (August 8) 1931, p. 390.

10. FOERSTER, H. R. Sporotrichosis, an Occupational Dermatosis, *J.A.M.A.* 87:1605 (November 13) 1926.

11. GOLDSMITH, W. NOEL: Recent Advances in Dermatology. Philadelphia, P. Blackiston's Sons & Co., Inc., 1936.

12. GRIETZ, O.: In *Handbuch der Haut und Geschlechtskrankheiten,* by Joseph Jadassohn, Berlin, Julius Springer, 1928. Vol. 11, p. 800.

13. HABERMANN, R.: In *Schadigungen D. Haut,* by Ullmann, Oppenheim and Rille, Vol. 2, pp. 447-468, Leipzig (Voss), 1926.

14. JORDAN, J. W., and WEIDMAN, F. O.: Coccidoidal Granuloma, *Arch. Dermat. & Syph.* 33:31-47 (January) 1936.

15. KINGERY and THIENES: Mycotic Paronychia and Dermatitis, A Hitherto Undescribed Condition Apparently Peculiar to Fruit Canneries, *Arch. Dermat. & Syph.* 11: 186, 1925.

16. LANE, C. G.: A Cutaneous Lesion Caused by a New Fungus (Phialophora Verrucosa), *J. Cutan. Dis.* 33:840, 1915.

17. LEVY-SERUGUE: *Gazette des Hospitaux,* 1906, p. 295.

18. MARKLEY, PHILPOTT and WEIDMAN: Deep Scopulariopsis of Ulcerating Granuloma Type Confirmed by Culture and Animal Inoculation, *Arch. Dermat. & Syph.* 33:627-641 (April) 1936.

19. MENSE, K.: Industrial and Cutaneous Changes Observed in the Tropics, in *Schädigungen d. Haut,* by Ullman, Oppenheim and Rille, Vol. 11, pp. 469-475, Leipzig, 1926.

20. MILIAN: Nouvelle Pratique Dermatologique, Vol. 4.

21. ORMSBY, OLIVER, S.: *Diseases of the Skin.* Philadelphia, Lea & Febiger, 1937.

22. OTA, M., and KOMAYA, G.: A New Species of the Genus Scopulariopsis (Bainter); S. Castelanii, *Dermat. Wchnschr.* 78:163, 1924.

23. RAVOGLI, A.: Blastomycosis of the Skin as an Industrial Disease, in *Schädigungen d. Haut,* Vol. 11, pp. 469-475, Leipzig, 1926.

24. ROETTER: *Calif. & West. Med.* 29:409-410, 1928.

25. SCHWARTZ, LOUIS I.: Chronic Paronychia, *Arch. Dermat. & Syph.* July, 1928, p. 74.

26. SERRA: *Gaz. degliospedali,* 1898, No. 8/8.

27. SUTHERLAND-CAMPBELL, H.: Paronychia, An Attempt to Prove the Etiological Factor in an Epidemic Among Orange Workers, *Arch. Dermat. & Syph.* Vol. 19, p. 33, February, 1929.

28. SUTHERLAND-CAMPBELL and PLUNKETT: *Idem.* Vol. 30, November, 1934.

29. TOMLINSON, C. C. and BANCROFT, PAUL: Granuloma Coccidoides, Report of a Case Responding Favorably to Antimony and Potassium Tartrate, *J.A.M.A.* 91:947-951 (September 29), 1928.

30. WHITE, R. PROSSER: The Dermatergoses or Occupational Affections, London, H. K. Lewis & Co., Ltd., 4th ed., 1934.

31. WILSON, HULSEY and WEIDMAN: Chromoblastomycosis in Texas, A Complete Histological and Mycological Study, *Arch. Dermat. & Syph.* 27:107, 1932.

32. Yearbook of Dermatology and Syphilology for 1935, p. 21.

33. *Idem.*, for 1936, pp. 40-41.

Lead Poisoning

—Section I. Exposures to Lead; Its Absorption, Transportation, Distribution, and Deposition in the Tissues; the Photometric Dithizone Method as an Accurate Means of Determination; "Normal" Lead in Tissues and Urine; "Normal" Value in Blood—

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DURING the past 10 years, experience in the Cook County (Illinois) Coroner's Laboratory indicates that lead poisoning is the most prevalent industrial disease in the highly industrialized area of Chicago, and that it is almost a daily occurrence that some physician or toxicologist is requested to express his opinion as to the possibility of lead poisoning in a given case.* There are approximately 4000 references on the subject in the scientific literature, but the absence of a correlation between the clinical and chemical findings has led to much confusion in the diagnosis of the disease, both before and after death. Because of this, those who are required to reach a decision often find themselves in a dilemma. There are no signs or symptoms which are pathognomonic of lead alone, and, therefore, the clinical evidence without reliable chemical findings is often insufficient for the purpose.

At the present time we know that traces of lead are present in all tissues and body fluids, at least in every one examined, as will be shown later. Therefore, the demonstration of the mere presence of lead is of no value as a diagnostic feature. It is necessary to show that lead is present in quantities which are of toxiocological significance. This has been done by correlating the concentration of lead in the respective tissues and fluids, with the history of exposure, observed signs and symptoms of intoxication, and anatomical findings.

Histories of the cases have been traced carefully by personal interviews with the victims in some cases, and through fellow-workmen, employers, relatives, attending physicians, hospital records, autopsy protocols, proceedings of coroner's inquests, and hearings before the Industrial Commission.

In order to arrive at the quantitative significance of lead in the tissues and body fluids, it was first necessary to determine the "normal" value in persons having no abnormal exposure and no symptoms of intoxication, and, second, to compare this value with that of individuals known to have had an abnormal exposure and definite symptoms of intoxication.

The photometric dithizone method was used for the determination of lead in all "normal" cases, and in all non-fatal cases, and in a great majority of the fatal cases. At the present time it is the most sensitive and accurate method for this purpose, and with a few simple modifications has been made specific for lead. The relative merits of various diagnostic features of the disease are discussed.

The treatment of lead poisoning has been the subject for much discussion, but the results of this investigation indicate that di-sodium phosphate, together with a normal calcium diet, appears to have the greatest therapeutic value.

Due to the lack of space it has been necessary to curtail many important details and references which may be found in the original paper. Only the most important findings and references having a direct bearing on the present work have been used.

An investigation of this type should be useful to medicolegal experts in reaching a decision as to what constitutes a case of chronic lead poisoning, especially those cases of an industrial nature.

Industrial Exposures

IN THIS district the greatest lead hazard is found among persons employed in the smelting and refining of lead, white lead factories, paint factories using lead pigments, the painting trade, storage battery plants, casket factories, automobile body assembly plants, and lead oxide plants.

The concentration of lead in the atmosphere bears a direct relation to the rate of plumbism. The inhalation of air contaminated with finely divided lead dusts or fumes is the most important mode of intoxication because absorption is greater and lead passes directly into the systemic circulation. Legge and Duckering[1] found no evidence of plumbism where employees breathed not more than 2 milligrams of lead during an eight-hour working day, equivalent to approximately 5 milligrams per 10 cubic meters of air if a man breathes 4.6 cubic meters in eight hours. The United States Public Health Service[2] considers the limit of safety for exposure to lead dusts or fumes to be not more than 1.5 milligrams per 10 cubic meters of air.

The daily swallowing of small amounts of lead results in absorption from the gastro-intestinal tract and subsequent intoxication. This is less dangerous than the inhalation of dusts and fumes because a very large percentage of lead is eliminated in the feces without ever having been absorbed.

The exposure of the intact skin to such a compound as tetraethyl lead results in absorption and intoxication.

Lead dusts are evidently composed of very fine particles of one or more of the following substances: metallic lead (Pb), lead peroxide (PbO_2), litharge or orange mineral (PbO), red lead (Pb_3O_4), lead chromate ($PbCrO_4$), lead arsenate ($PbH_4(AsO_4)_2$), the white leads as the basic car-

* An abstract of a thesis submitted in partial fulfillment of the requirements for the Degree of Doctor of Philosophy in Pharmacology in the Graduate School of the University of Illinois, 1938.

bonate ($2PbCO_3.Pb(OH)_2$), and the basic sulfate ($PbSO_4.PbO$). The atmosphere becomes contaminated with the dusts by mechanical agitation, by air currents, or by both. Lead fumes are apparently due to the volatilization by heat of the lower oxides.

Non-Industrial Exposures

SIX out of 10 fatal cases of poisoning in children reported in this paper were caused by "pica," a depraved appetite or unnatural desire to eat dirt or gnaw on unnatural articles of food, which articles were metallic lead toys, and porch railings, cribs, window-sills, etc., covered with paints containing lead. The other four cases were due to dust and fumes resulting from burning old storage battery cases as fuel.

Drinking water may be a source of lead poisoning if allowed to stand in lead pipes as reported by Quan and Klein[3], and several such epidemics have been reported.[4] The same would be true of beer or any beverage or food cooked or allowed to stand in contact with lead, particularly when carbon dioxide is present.

The United States Food and Drug Administration closely guards the lead content of food, the limit being two parts per million, or one one-hundredth of a grain per pound. This is of the utmost importance because many vegetables and fruits, especially apples, are sprayed with lead-arsenate.

Lead poisoning in infants whose mothers used cosmetics and hair dyes containing lead was reported by Dufour,[5] Suzuki and Kaneko,[6] Fukushima and Matsumoto[7]; from rubber nipples containing lead, and lead nipple shields by Lewin,[8] and Campaner.[9]

Plumbism due to lead foil wrappings of snuff was found by Uttal,[10] and by Bauer and Ropes.[11]

A case of acute lead poisoning is reported in this paper where medicine containing lead (supposedly lead acetate) was taken by mistake.

Absorption Through the Respiratory Tract

IT IS a generally accepted fact that the absorption of lead through the respiratory tract is the most dangerous avenue of intoxication. The inhalation of lead dust is extremely hazardous because it has but one way out — expulsion by cilia. That which enters the bronchioles and alveoli apparently remains there until absorbed, and enters directly into the systemic circulation. Lead is absorbed by being dissolved in the serum and by phagocytosis. Finely divided particles of metallic lead have been found to be "less readily phagocytized" than lead compounds, and it is believed that the irritant action of unphagocytized material in the lungs may lead to a proliferation of fibrous tissue and predispose to tuberculosis which is so common among printers. An equivalent amount of lead is 10 to 100 times more toxic when inhaled than when swallowed. These facts have been successfully demonstrated in the experimental works of Legge and Goadby,[12] Minot,[13] Blumgart,[14] Fairhall,[15] Fine,[16] and Drinker.[17]

Absorption From the Gastro-Intestinal Tract

LEAD poisoning in the six children obsessed with "pica" reported here, and many other cases resulting from the imbibing of water, beverages, food and medicine contaminated with lead, are sufficient proof that lead is absorbed from the gastro-intestinal tract.

Carlson and Woelfel[18] found that the white leads were very soluble in human gastric juice, running parallel to the solubility in 0.5% hydrochloric acid, and depending upon the percent of free hydrochloric acid in the stomach. When gastric juice was mixed with milk in sufficient quantity to fix all of the free acid by the milk proteins, or neutralize it by the alkali salts, the solubility of lead was diminished. Carlson advocated the use of milk as a prophylactic measure in preventing lead absorption because, as he states, "there is less appetite secretion of gastric juice with milk" in comparison to other forms of protein, "and the fat in the milk depresses and retards the action of gastric secretagogues." His opinion "that the digestive tract is in some cases the sole, and probably in all cases the most important avenue of absorption of lead in industrial poisoning" cannot be accepted in view of the more recent findings of Minot, Blumgart, and Goadby.

Aub and his colleagues[19] were of the opinion that the larger portion passed into the colon and out through the feces, a small part being absorbed in the small bowel, carried to the liver through the portal circulation, and from there through the systemic circulation to the viscera and central nervous system; that some of the lead in the liver is re-excreted by the bile into the small intestine, and may not have reached the general circulation at all. Lead which has reached the systemic circulation is excreted in the urine by the kidneys, by the liver through the bile, and some is carried directly to the intestines by the general circulation.

Absorption Through the Intact Skin

APPARENTLY no one has been able to present any concrete examples and conclusive evidence that lead can be absorbed through the unbroken skin under ordinary working conditions, other than organic lead compounds such as tetraethyl lead, or preparations of lead in oil. Numerous cases are recorded where lead has been absorbed through the broken skin, such as preparations applied to burns, ulcers and eruptions. Absorption may occur through delicate mucous membranes when lead preparations are used as vaginal douches and eye washes.

Distribution in the Tissues

IN THE examination of the various soft tissues and bone from 75 autopsies on human beings, it has been the writer's experience that lead was detectable in each tissue in every case. The concentration (milligrams of lead per 100 grams of tissue) ranked first in the bone, second in the liver, and third in the kidney, almost invariably. The only exceptions to this were a few cases where

death occurred so soon after exposure that the elapsed time was insufficient for a greater deposition of lead in the bone. It is unfortunate that a wider variety of specimens was not obtained but experience indicates that liver, kidney and bone are usually sufficient. The work on bone has had to be confined largely to sternum and ribs due to certain limitations on autopsies in this community. Tompsett[20] has found a much higher concentration in long bones (femur and tibia). The presence of lead in all tissues examined, and the distributions are in agreement with the work of Tompsett and Anderson.[21]

Transportation

IF WE are to believe the theory and work of Aub and co-workers,[19] lead is not precipitated in the blood as the phosphate but is transported largely as a finely divided colloidal lead phosphate ($PbHPO_4$). They state that it is converted into this form by the peptizing effect of the body serums and demonstrated this by conclusive experiments. We must not expect all of it to be in the form of the phosphate because even it is soluble to a certain extent. Apparently the toxicity is due to small amounts of soluble salts, but the theory that lead is transported in the blood stream largely as the colloidal lead phosphate is the more tenable. The existence of the phosphate and more soluble salts depends upon several factors such as the ratio of various anions to cations, O_2 tension, and hydrogen-ion concentration. Absorption, deposition, reabsorption and excretion must proceed at an increased rate to the point where equilibrium is established, and then decrease.

A point in favor of the transportation of lead in the blood stream as the colloidal phosphate is the fact that in most cases of chronic lead poisoning there has been an exposure to the more soluble forms of lead (metallic lead and its compounds other than the phosphate). The time required for the appearance of toxic symptoms varies from several weeks to several years, depending upon the severity of exposure. If lead were transported in the blood and deposited in the soft tissues as the more soluble and toxic forms we would expect toxic symptoms to appear almost immediately, or at least much more quickly than they actually do. Therefore, it appears that the conversion of the more soluble forms of absorbed lead into the more insoluble and relatively non-toxic phosphate is one of the means by which the body combats the toxicity of lead but the question is largely academic.

Deposition in the Tissues

IT IS generally accepted that the metabolism of lead follows closely that of calcium, and that most of it is deposited in bone as the tertiary phosphate [$Pb_3(PO_4)_2$], its most insoluble form. Aub and co-workers[19] found that most of the lead was deposited in the intracellular spaces, the trabeculae, and compact material of the shafts, and not in the organic matrix; that the maximum deposition occurred between pH 7.4-7.8, above or below which it was mobilized. The fact that the normal pH of blood is about 7.3 accounts for the remarkable storage of lead in bone, and that it may be liberated by an acidosis or alkalosis.

Hunter and Aub[23] administered parathyroid extract to patients suffering with chronic lead poisoning, and observed that the excretion of lead was definitely increased when the extract was first given, but subsequent administrations produced little or no increase. They explain this by concluding that lead excreted after the first doses is that which is loosely bound in the bones or has been recently ingested, calling it "readily available." By the same procedure the excretion of calcium was increased.

Aub, Robb, and Rossmeisl[24] later found that most of the lead in bone, particularly in relation to calcium, was found in the trabeculae and not in the cortex. The trabeculae act as a storehouse for available calcium and lead stored here was also assumed to be available and more easily mobilized. By keeping leaded animals on a low calcium diet they were able to show that the trabeculae disappeared and lead decreased. As calcium was needed it was drawn from the trabeculae, and lead was mobilized also.

In the present work 52% more lead was found in the cortex than in the trabeculae of the sternum in an individual known not to be suffering from lead poisoning (Case 45, Table XII), but the reverse was true in an active case of chronic lead poisoning. Seventy-eight percent more lead was found in the trabeculae than in the cortex in Case 25, Table X, indicating that most of the lead in this case was available. It seems that recently ingested lead is first deposited as a loosely bound compound in the trabeculae, in which form it is easily liberated, thus supplying a constant stream to the tissues through the blood. In time some of it is pushed away from the blood and deposited as a compact mass of insoluble tertiary phosphate in the cortex, from which it is less readily liberated and remains deposited for an indefinite period. Apparently trabecular lead is of greater significance than cortical lead in chronic lead poisoning.

Lead is apparently deposited in the soft tissues largely as the relatively more soluble proteinate, as compared to the phosphate, but other forms must exist in less quantity than the proteinate. In the presence of various cations, anions, organic acid radicles, and changes in pH, it is impossible to predict what the tissue picture would be. Microscopic examinations of sections of tissues from well established cases of lead poisoning here by the late Dr. Richard H. Jaffé, Chief Pathologist of the Cook County Hospital, have never exhibited any tiny opaque particles of metallic lead.

The Photometric Dithizone Method

DITHIZONE (diphenylthiocarbazone, C_6H_5NH $NHCSN{=}NC_6H_5$) was first introduced by H. Fischer[25] in 1929 for the detection of heavy metals, and was applied to the determination of lead by Fischer and Leopoldi[26] in 1933. When dissolved in chloroform or carbon tetrachloride the dye imparts a bluish-green color to the solution which, when shaken with an ammoniacal cyanide solu-

tion of a small amount of lead, turns red due to the formation of lead dithizonate. According to Fischer, two molecules of dye react with one atom of lead. Lead dithizonate is soluble in chloroform and carbon tetrachloride, and this principle has proved to be of great value in the separation of lead from other metallic elements and extraneous salts.

An excellent criticism of the various methods formerly used for the determination of small amounts of lead may be found in the work of Wichman, Murray, Harris, Clifford, Loughrey, and Vorhes.[27] The method of Fairhall[28] involving the precipitation of lead first as the sulfide, then as the chromate, with the subsequent iodimetric titration, was probably the most widely used until the advent of dithizone. The disadvantages of the Fairhall method seem to outweigh the advantage that it is specific for lead, unless a large amount is present. It is very tedious, time consuming, and apparently involves an inherent loss of lead. During the early part of the present investigation the Fairhall method was used with the result that in many instances where the exposure to lead was slight, a negative or doubtful result was obtained on specimens of urine and tissues, and due to this fact these cases have been excluded. Such has never been the case since the dithizone method has been used in the determination of lead in more than 200 specimens of soft tissue and bone, and approximately the same number of urine and blood samples. Aub, Fairhall, Minot and Reznikoff[19] report no lead in many of the specimens examined in Table 18, page 71, of their monograph. This criticism should not be misconstrued. If lead is present in toxicologically significant quantities it can be determined by Fairhall's method, and in spite of the inherent loss or lack of sensitivity will still come within the toxic range, if a sufficiently large sample is used. However, if the lead content of the tissues is exceptionally low, as in the "normal" and "unjustified" groups of the present paper, the method will sometimes yield a negative result, and at other times an uncertain one. The analyst who is familiar with the vagueness of the starch-iodide end-point which is encountered when extremely small amounts of free iodine are titrated with 0.005 normal sodium thiosulfate, will agree that the accuracy is not greater than 0.1 of a cubic centimeter of the thiosulfate, equivalent to 0.0345 mgm. of lead.

Clifford and Wichman[29] have discussed the dithizone "titrimetric-extraction" and "one-color" methods, and the disadvantages mentioned by them have been confirmed in the present work. Various modifications have been published by Tompsett and Anderson,[21] Fischer and Leopoldi,[26] Wilkins, Willoughby, Kraemer, and Smith,[30] Horwitt and Cowgill,[31] Seelkopf and Taeger,[32] Winter, Robinson, Lamb and Miller,[33] and Ross and Lucas.[34] The present writer was unable to confirm the findings of Horwitt and Cowgill,[31] that excess dithizone and bismuth may be stripped from the dithizone complex by shaking with 1.5 volumes of 0.5% potassium cyanide for 10 seconds, with no significant loss of lead.

The "mixed color" method proposed by Clifford and Wichman[29] has been found to be the most reliable. With a simple modification it has been made specific for lead.

The lead is extracted with an excess of dithizone in chloroform but the excess is partitioned between the chloroform and aqueous phase. The color of the chloroform layer depends upon the quantity of lead present and excess dithizone, and varies from the blue-green of dithizone to the red of lead dithizonate, with intermediate shades of crimsons, purples, and blues. The term "mixed-color" is derived from the blending of the red and green. Clifford and Wichman[29] state that "if the extraction is made under definite conditions of volume and strength of dithizone solution and volume and pH of aqueous fraction, the "mixed-color" obtained is definite and reproducible, and, provided excess dithizone is present, depends only upon the amount of lead present." This is true if other contaminating metals, principally bismuth, have been removed.

The color may be matched by a visual color comparison in Nessler tubes or vials against prepared standards. This involves the preparation of a series of standards at the same time a determination is being made. A hydrogen-ion colorimeter may be used but exact color matches are difficult to obtain, and there is an unavoidable evaporation of chloroform from the cups. The photelometer (similar to a photometer with photo-electric cell) eliminates any personal factor but the photo-electric cell must be non fatigueable and the electric current constant.

The photometer used in this work is essentially the same as that described by Clifford and Wichman,[29] who kindly submitted instructions for its construction, but improvements were made. Numerous wire connections and light sockets were eliminated by using two 250-watt projection bulbs as the light source. Better insulation against heat was provided for the neutral density measuring wedge by constructing the light box of Bakelite board and boring holes for ventilation in each end near the top.

The method of analysis is essentially the same as that proposed by these writers with two exceptions: (1) the sulfide precipitation of lead in bone was found to be unnecessary, and (2) 1-1000 nitric acid solution (pH 2.0) was used in stripping lead from dithizone instead of 1-100 acid because in this way bismuth was more easily eliminated.

INTERFERENCES. Dithizone is not specific for lead because a solution of it in chloroform or carbon tetrachloride will form colored complexes with zinc, cobalt, nickel, cadmium, silver, copper, mercury, stannous tin, bismuth, thallous thallium, and lead. The presence of excess potassium cyanide prevents the extraction of all but the last four. Traces of tin gain entrance to the body through food and drink. Thallium is used in certain German rat pastes, but its occurrence in biological specimens in this district is very rare. Both stannous tin and thallous thallium are converted to stannic tin and thallic thallium by ashing, evaporating with nitric acid to further oxidize, and re-

ashing, thereby eliminating the possibility of their extraction in reduced form. The fact that bismuth preparations are frequently administered therapeutically, and that much of it will be extracted along with the lead, is the reason why bismuth in particular must be guarded against.

ELIMINATION OF BISMUTH. Willoughby, Wilkins, and Kraemer,[35] and later Hubbard,[36] eliminated bismuth by adjusting the acid stripping solution to pH 2.0 with dilute ammonia and extracting bismuth with a large excess of dithizone. Horwitt and Cowgill[31] suggested that a pH between 3.0 and 3.5 be used.

Since bismuth is extractable with difficulty at pH 2.0, an attempt was made to find the optimum pH. Standard solutions of bismuth and lead were made up in dilute nitric acid solutions having the following pH: 2.0, 2.5, 3.0, 3.5, and 4.0 as determined by a pH electrometer. Measured quantities of each solution were added to separatory funnels and the volume adjusted to 50 cc. with the proper strength of acid. Repeated extractions with excess dithizone were made until there was no further perceptible color change, showing that all bismuth had been removed. The pH was then adjusted to 9.5 with ammonia-cyanide mixture, lead was extracted with the proper strength and volume of standardized dithizone, and the final estinations were made photometrically. As the pH was increased from 2.0 up to and including 3.5 the extraction of bismuth was facilitated and all lead was recovered. At pH 4.0 the extraction of bismuth was retarded and much of the lead was lost. Best results were obtained by shaking for two minutes. Under these conditions the optimum pH appeared to be 3.5. This indicated that lead should be stripped from the combined original dithizone extracts with acid of this strength, but the results were very unsatisfactory.

Measured amounts of lead and bismuth were added to the citrated ammoniacal-cyanide solution (as described later in the method), and extracted with excess dithizone. The combined extracts were stripped in some instances with 50 cc., and in others with 100 cc. of dilute nitric acid pH 3.5, but the dithizone showed very little tendency to revert to the original blue-green color. This indicated that the bismuth complex was being retained in the chloroform, and that very little lead was partitioning into the acid-aqueous phase. The subsequent estimation of lead in the aqueous solution revealed that most of it had been lost.

These results indicate that lead, in the presence of excess dithizone in the chloroform phase will not partition quantitatively into an aqueous phase of acid pH 3.5. It seems that the mass-action effect of excess dithizone in chloroform when lead is contained therein is far greater than the mass-action that an acid solution of pH 3.5 might exert in breaking the complex.

Stripping with 1 per cent nitric acid solution (1 cc. in 100) is entirely satisfactory if bismuth is absent, but becomes unnecessarily difficult if present because practically all of the bismuth will partition into the aqueous phase along with the lead, thereby increasing the difficulty of removing bismuth at pH 2.0. This can be overcome to a very large extent by stripping with an aqueous acid solution of pH 2.0.

When 1 cc. of concentrated nitric acid is made up to 1000 with redistilled water, the pH is 2.0-2.05 as determined by a pH electrometer. Acid of this strength will strip more than 200 gamma of lead from the dithizone complex, but will extract only part of the bismuth, if present. A procedure in which an aqueous-acid stripping solution of pH 2.0 is used has the following advantages: (a) only part of the bismuth present in the first dithizone extract is partitioned into the aqueous phase; (b) an extra operation involving the adjustment to pH 2.0 is saved because the acid already has the desired pH. In this way bismuth and also any stannous tin present is eliminated in three steps in the method: (1) the high pH and cyanide of the original citrated-ammoniacal solution retards the extraction of bismuth to a certain degree; (2) stripping the combined dithizone extracts with acid of pH 2.0 allows only part of it to partition into the aqueous phase; and (3) that portion which enters the acid solution is removed by repeated extractions with excess dithizone. Thus, while no further work is involved, the method becomes specific for lead.

Apparatus and Materials

ALL water should be redistilled in an all-glass Pyrex distilling apparatus. Filter papers—use Whatman No. 4 or a similar grade, 9 or 11 cm. diameter. They are much cheaper than No. 42 and filter more rapidly, thus decreasing the evaporation of chloroform when the final dithizone extracts of lead are filtered. Filter papers should be soaked over night in 1% HNO_3 (1-100), and washed free of all traces of lead and acid with a large volume of redistilled water on a Büchner funnel with suction. They should be dried over sulfuric acid in a vacuum desiccator. Do not attempt to dry in an oven because the paper becomes brittle and cannot be folded without breaking. All glassware should be cleaned with warm 10% NaOH, rinsed with water, then with hot 50% HNO_3, and finally rinsed thoroughly with redistilled water. All reagents should be stored in glass-stoppered Pyrex bottles with bottle caps, but the Squibb separatory funnels may be of Kimble glass which has been found comparable to Pyrex in avoiding lead contamination. It is advisable to clean the separatory funnels immediately before use by shaking out with a mixture of 50 cc. of 1-1000 HNO_3 10 cc. of the ammonia-cyanide mixture (described later), and a few cc. of dithizone solution, and then rinse thoroughly with redistilled water. Glass stoppers are tied on with chromel wire (28 gauge).

Standard dithizone solutions should be dispensed from automatic burettes, made entirely of amber glass, with a ground glass joint between the burette and reservoir. The reservoirs are preferably of 2,000 cc. capacity. The amber glass excludes ultraviolet rays which decompose the dye. In practice only two are usually necessary—one for the 0-200 gamma lead range used in the pre-

liminary extraction, and another for the 0-20 and 0.50 range, which ranges require the same strength but different volumes of dithizone.

Ashing dishes are Sillimanite evaporating dishes (with lip) of 175 cc. capacity, cleaned and rendered free of any surface lead in the same manner as the glassware. Silica dishes are to be preferred because of the freedom from lead and higher fusion temperatures with alkaline ashes. Refractory substances in the ash are broken up by repeated evaporation with acid and subsequent dehydration. Fusion of the glaze is prevented to a large extent by placing the dish and contents in a cold electric muffle furnace and so regulating the rheostat that the temperature never exceeds 500° C. As soon as any etching of the glaze is noticed the dish should be discarded. In this way loss of lead is prevented when Sillimanite dishes are used.

Use a large glass-stoppered Pyrex bottle for the storage of redistilled water, and Pyrex wash-bottles with ground glass joints for water and chloroform.

Lubricate all stop-cocks with a good grade of white petrolatum.

Reagents

HYDROCHLORIC acid — C. P. concentrated reagent, sp. gr. 1.18-1.19, containing not more than 0.0005% heavy metals as lead.

(b) Nitric acid—C. P. concentrated reagent, sp. gr. 1.42-1.425, 70-71% HNO_3, containing not more than 0.00002% heavy metals as lead.

(c) 0.1% Nitric acid (pH 2.0)Make 1 cc. of reagent (b) to 1,000 cc. with redistilled water.

(d) Ammonium hydroxide—C. P. concentrated reagent, sp. gr. 0.90, containing not more than 0.0000% heavy metals as lead.

(e) 10% Potassium cyanide solution—Dissolve 50 grams of potassium cyanide (reagent grade) in redistilled water and make up to 500 cc.

(f) Ammonia-cyanide mixture—A mixture of 7.5 cc. of reagent (d) and 10 cc. of solution (e) is made up to 500 cc. with redistilled water.

(g) 50% Citric acid solution—Use the "low lead analytical reagent grade" containing not more than 0.00005% heavy metals as lead. Dissolve 500 grams in redistilled water, and make up to 1,000 cc.

(h) Chloroform—Redistill the U.S.P. grade in an all-glass Pyrex distilling apparatus. Traces of water are removed by allowing the distillate to drop directly on a lead-free filter paper inserted in the mouth of a glass-stoppered lead-free amber glass bottle used for storage.

(i) Purified dithizone (dry)—The commercial product is only about 85% pure and contains certain sulfur compounds and chloroform insoluble material. Dissolve 5 grams in 200 cc. of chloroform in a large separatory funnel. Add about 500 cc. of 5% ammonia (5 cc. in 100) and shake. The impurities are left in the chloroform layer which is discarded. Wash with a few cc. of chloroform and discard the washings. Precipitate the dye in the aqueous solution by adding 10% hydrochloric acid (10 cc. in 100) drop by drop until precipitation is complete. Repeatedly extract the dye with small portions of chloroform until extraction is complete. Combine the extracts in a Pyrex evaporating dish and evaporate the chloroform on a very slow steam-bath. Dry to constant weight over recently ignited lime in a vacuum desiccator, and store in a small glass-stoppered bottle of amber glass.

(j) Standard dithizone solutions—dissolve 125 mgm. of (i) in chloroform in a 250 cc. volumetric flask and make up to volume with chloroform. Each cc. is equivalent to 0.5 mgm.dithizone. Use this solution for making the other standard dithizone solutions. It is measured from a burette (reading from top of meniscus) into a 1-liter volumetric flask and made up to volume with chloroform for the various ranges as follows:

1. 0-5 and 0-10 gamma lead range, 8 cc. per liter.
2. 0-20 and 0.50 gamma lead range, 16 cc. per liter.
3. 0-100 gamma lead range, 32 cc. per liter.
4. 0-200 gamma lead range, 40 cc. per liter.

In practice (1) is usually made up only as needed. It is used at infrequent intervals for exceptionally low ranges and reagent blanks. (3) may also be prepared as needed. (2) and (4) are prepared and stored in the burette reservoirs protected from sunlight. (4) is used for the original extraction of lead, and the volume used indicates the range. (2) is for the final estimation of lead, unless the indicated range is higher or lower.

(k) Standard lead solutions—Use lead nitrate [$Pb(NO_3)_2$, special reagent grade, in which the maximum limit of total impurities does not exceed 0.002%]. Weigh 1.5985 gm. of this into a 1-liter volumetric flask, add 10 cc. of concentrated nitric acid and a few cc. of redistilled water to dissolve, make up to 1000 cc. with water, and mix. This solution, which contains 1 mgm. of lead per cc., is quite stable and is used as the "standard lead stock solution." It should be discarded if any cloud or sediment is observed, but this is unlikely.

(l) Standard lead solution—1 cc. contains 0.010 mgm. Pb. Dilute 100 cc. of stock solution to 1000 with redistilled water, and then dilute 50 cc. of this to 500 with 1-1000 nitric acid. Use in standardizing dithizone for the 0-100 and 0-200 gamma lead ranges.

(2) Standard lead solution—1 cc. contains 0.001 mgm. Pb. Dilute 50 cc. of (1) to 500 cc. with 1-1000 nitric acid. Use in standardization of 0-5, 0-10, 0-20, and 0-50 ranges. Make up (1) and (2) as needed because lead will separate out from the weak acid solutions.

(L) Acid metacresol purple indicator—0.04% solution in redistilled water.

Standardization of Dithizone

IT IS best to make readings on the scale of the photometer at several points in each range of lead. Any change in the dithizone can be detected by checking the zero lead reading about once a week. The concentrations and volumes of dithizone, together with the cell lengths, for each respective range of lead are found in Table I.

From a burette deliver the required amounts of standard lead solution into a series of 125 cc. sep-

aratory funnels. Add enough reagent (c) to bring the volume to 50 cc. For 0 lead use 50 cc. of (c). Saturate each solution with chloroform by shaking with 2-3 cc. Let stand a few minutes and shake down any globules of chloroform on the surface of the liquid or clinging to the sides. Draw off completely the chloroform layer, being careful not to lose any of the aqueous solution. Stopper the funnel and shake chloroform from the stem. Invert and open the stopcock gradually blowing back any droplets of aqueous solution into the funnel. Add 10 cc. of solution (f) and mix. From the burette add the specified volume of dithizone and shake for one minute. Let stand two minutes and then filter the chloroform extract into a dry 50 cc.

TABLE I
REQUIRED DITHIZONE AND CELL LENGTHS FOR VARIOUS LEAD RANGES

Lead Ranges (gamma[a])	Dithizone Concentration (mgm./l.)	Dithizone Content (mgm.)	Volume (cc.)	Cell Length (inches)
0-5	4	0.02	5	2[b]
0-10	4	0.04	10	2
0-20	8	0.08	10	1
0-50	8	0.20	25	1
0-100	16	0.40	25	½
0-200	20	0.60	30	½

a-0.001 mgm.
b—Special cell, 3.5-4.0 cc. capacity.

Florence flask of quartz or Pyrex. The use of a funnel is avoided by folding the lead-free filter paper and inserting gently into the neck of the flask, which is preferable to a small beaker because there is less evaporation of $CHCl_3$. Fill the proper absorption cell almost to the top of the vent, after first rinsing with some of the extract, and set in trough of the photometer. Take the average of five or 10 readings, most of which are usually the same and rarely vary more than 2 mm. Plot scale readings against gamma of lead on a large scale graph.

Procedure

URINE—Measure 100 cc. of thoroughly mixed urine into an evaporating dish and bring to dryness on the steam bath.

BLOOD—Measure the volume of blood in the syringe with which it was drawn and expel it into the evaporating dish, if it is convenient to do so. Evaporate to dryness on the steam bath. Otherwise, the sample must be placed in an oxalated blood bottle and pipetted into the dish. The bottle should contain a known quantity of the oxalate, the lead content of which is known.

SOFT TISSUES (liver, kidney, etc.) — Strip the specimen of any fat or other adhering tissue. Place on a clean board and make several cross sections of the organ with a clean steel knife. Mince finely and weigh 20 grams into the evaporating dish. Weighings should be made on an analytical balance to the nearest 0.1 gram. Dry for a short time on the steam bath.

BONE—Strip clean of all adhering tissue and cut several small cross sections, by means of a clean chisel and hammer. Weigh 5 grams into the dish on an analytical balance.

Place in a cold temperature-controlled electric muffle furnace, and gradually bring the temperature up to 500° C. Ash over night at this temperature. A light gray ash is obtained, and there is no spattering or fusion. Remove the dish, cool, add 2 cc. of concentrated HNO_3, and evaporate to dryness on the steam bath. Replace in the muffle and re-ash for about 30 minutes or until a pure white ash results.

Remove the dish, cool, add 10 cc. of the concentrated HCl, and bring to dryness on the steam bath. Add another 10 cc. portion of HCl and again evaporate to dryness, to decompose traces of silicates and other refractory salts. Remove the dish, carefully wipe off the bottom, and while it is still hot add 2 cc. of HCl and about 20 cc. of hot water to completely dissolve the ash. Pour into a 250 cc. separatory funnel. Add 10 cc. of 50% citric acid solution (g) to the dish, a small amount of hot water and swirl gently before adding the contents to the separatory funnel. Use 20 cc. or more of 50% citric acid for bone. Rinse the dish thoroughly three times with hot water. Mix the contents of the separatory funnel and add two or three drops of acid meta cresol purple solution (L). Adjust to pH 8.5 with ammonia (d) from a burette (usually 7-8 cc.) and cool. Add 5 cc. of 10% KCn (e) to the dish, rinse into the separatory funnel with water and mix. The total volume should be 100-125 cc.

Extract the lead by adding dithizone solution (j4) from the automatic burette, 5 cc. portions at a time, shaking between additions until the chloroform extract assumes a purple color. Let stand a few minutes and shake down globules of chloroform. Draw off the chloroform layer (leaving a $CHCl_3$ trap in the stop cock) into a 125 cc. separatory funnel containing 50 cc. of 1-1000 nitric acid solution (pH 2.0). Repeat the extraction of the aqueous solution with 20 cc. of the dithizone, or a smaller portion if very little lead is present, and combine the chloroform extracts.

Shake for one minute to strip lead from the dithizone complex. Discard all but 2 or 3 cc. of the dithizone, dilute with 2-3 cc. of $CHCl_3$, and shake for two minutes. If the dithizone retains its original blue-green color, bismuth is absent. A dirty purple or iridescent blue indicates a trace of bismuth, and yellowish-brown a larger amount. If present, bismuth must be extracted repeatedly with excess dithizone, shaking for two minutes. Its removal is complete when 2 or 3 cc. of dithizone retains its original color after shaking for two minutes. Discard the dithizone layer and wash the aqueous solution with 2-3 cc. portions of $CHCl_3$ until free from dithizone. Shake down globules of $CHCl_3$ and allow to separate for a few minutes. Draw off $CHCl_3$ completely. Stopper the funnel, invert, open stop cock, and blow back any droplets of aqueous solution in the stem.

Add 10 cc. of ammonia-cyanide mixture (f) and mix. Add the required amount of standardized dithizone as found in Table I, and shake for one minute. Let stand two minutes and filter through a folded lead-free filter paper inserted in the neck of the small Florence flask. Rinse out the proper absorption cell with a small amount of the extract,

and then fill it almost to the top. Take the readings immediately. Calculate the amount of lead present by referring to the prepared graph for the particular range of lead.

For the convenience of the analyst who is unaccustomed to the method, the following information will be useful. In the initial extraction of lead (bismuth absent) where dithizone (j4) is added in 5 cc. portions, the lead range is indicated by the following colors:

0- 5 if 5 cc. of dithizone is green;
0- 10 if 5 cc. of dithizone is blue;
0- 20 if 5 cc. of dithizone is purple, but not red;
0- 50 if 10 cc. of dithizone is purple;
0-100 if 20 cc. of dithizone is purple;
0-200 if 30 cc. of dithizone is purple.

If the range exceeds 200 gamma, strip an aliquot of the combined initial dithizone extracts or, better, use an aliquot of the nitric acid stripping solution.

Reagent Blanks

TWO lead blank determinations are necessary—one for urine, blood and soft tissues, and another for bone in which the only difference is 20 cc. of citric acid instead of 10 cc. The blanks are run in exactly the same way as the actual lead determinations. A new blank is required with each new batch of reagents, or if the reagents are stored for a long period, which increases the lead contamination from the glassware. Dirt must be prevented from falling into the dishes by thoroughly cleaning the muffle furnace and covering the steam bath. Dust must be excluded from the laboratory as far as possible.

When the lead-range is so high that it becomes necessary to use an aliquot of the total extract, only a corresponding part of the total blank should be subtracted from the lead found in the aliquot.

In the present work the blanks varied from 0.5 to 1.9 gamma of lead.

Sensitivity

WHEN a graph is plotted for the 0-5 gamma lead range it can be seen that each millimeter of the scale corresponds to approximately 0.07 gamma, equivalent to 0.00007 milligram or 0.00000007 gram of lead. This indicates that as little as seven parts of lead in 100,000,000 may be determined accurately if the blank is reduced to hundredths of a gamma. Since it has not been possible to obtain blanks less than tenths of a gamma, it is not practical to estimate lead closer than the nearest hundredth of a gamma in the lower ranges, and the nearest tenth in the higher. The method is so sensitive that the writer was able to determine lead in the fabric surrounding a bullet hole in a coat, in connection with a murder case.

Accuracy

PROVIDING the necessary precautions are followed, the accuracy of the method depends upon the skill and technique of the analyst. It is necessary that the reagent blanks be kept as low as possible, especially when working with exceedingly low ranges of lead. Ferric iron in the presence of cyanide will oxidize and destroy the color of dithizone. This difficulty is encountered with specimens which are high in iron content such as blood and liver. The disappearance of color might lead the chemist to believe that lead is absent, and the destruction of dithizone will prevent the extraction of lead. To overcome this, the pH should be about 9.5 and the mixture is shaken gently for a few seconds and stopped at the moment it begins to fade. Repeated additions of excess dithizone with gentle shaking for about 10 seconds will extract all of the lead. Wichman[27] suggested that less cyanide be used, and Fischer[25] that hydroxylaine hydrochloride be added to prevent the oxidation. Horwitt[31] obtained the best results by using less cyanide and also adding hydroxylamine hydrochloride. It is best not to use less cyanide because

TABLE II

RECOVERY OF LEAD

Specimen	Pb present originally (gamma)	Pb Added (gamma)	Bi, Sn(ous), Zn, Tl(ous), As, added (gamma)	Total Pb found (gamma)	Recovery of added Pb (per cent)
Liver (20 gm.)	48.5	50		98.7	100.4
"	21.0	100	10 of each	118.0	97.0
"	25.0	20		45.0	100.0
"	42.5	200		241.0	99.3
Kidney (20 gm.)	20.0	50		70.0	100.0
"	14.8	100	10 of each	114.0	99.2
"	13.4	20		33.2	99.0
"	22.5	200		223.6	100.1
Bone (5 gm.)	53.8	50		103.0	98.4
"	47.3	100	10 of each	148.0	100.7
"	27.5	20		47.5	100.0
"	85.0	200		280.0	97.5
Blood (20 cc.)	9.0	50		58.8	99.6
"	4.0	10		14.1	101.0
"	11.0	20	5 of each	31.3	101.5
Urine (100 cc.)	29.5	50	10 of each	79.5	100.0
"	3.0	10		13.0	100.0
"	1.9	20		21.7	99.0
"	5.0	100		105.6	100.6

the chloroform extracts might become unnecessarily contaminated with other metals. Hydroxylamine hydrochloride will prevent oxidation to a large extent, and can do no harm other than possibly increase the blank. In the present method, the oxidizing effect of ferric iron and cyanide on dithizone is of little consequence because the first extraction is preliminary to the final estimation. The volatilization of tin as $SnBr_4$ is not necessary in working on biological specimens, as indicated in the recoveries obtained, unless unusually large amounts are suspected. The accuracy of the method is illustrated in Table II where known amounts of lead and other metallic substances were added to specimens of known lead content. The recovery of added lead varied from 97 to 101.5%, the average being 99.1%. These results indicate that the method is accurate to ±1.0%.

The "Normal" Occurrence of Lead in Tissues of the Human Body

—25 Cases—

SO FAR as is known, no one has been able to show that traces of lead are necessary for the maintenance of life, or that they have any particular vital function. With the development of photometric and spectrographic methods it has been possible to demonstrate that lead is present in varying small amounts in each human tissue examined. These traces of lead which are not known to have any demonstrable good or bad effect on the human organism may be termed "normal" because they occur in all cases regardless of age or exposure to lead. The source is in food, drink, and atmosphere that has been contaminated with small amounts of lead. Kehoe and his co-workers[37 to 40] made an extensive investigation of the normal absorption and excretion of lead, and the sources of normal lead absorption. As a result of their exhaustive study there can be no doubt that food, drink, and atmosphere are the chief sources of "normal" lead. Granting that most foods contain less than two parts per million of lead, or one one-hundredth of a grain per pound (limit set by Food and Drug Administration), and that the air which we normally breathe contains less than 1.5 mgm. of lead per 10 cubic meters, it follows that these and drink must be the source of the traces found in all normal human beings. Exposures other than these, or those occurring otherwise than under ordinary normal living or working conditions may be considered abnormal. The above limits have been found to be harmless, mainly because most of the lead entering the body is eliminated as such without having been absorbed. The small percentage that is absorbed is the source of "normal" lead. There is no reason to believe that it should be present normally, or that it exerts any necessary function. The constant exposure to higher concentrations results in a greater absorption and in chronic lead poisoning. It is possible that an individual may have suffered the effects of chronic lead poisoning at some time, and recovered. The recovery process consists of the elimination of lead through the feces and urine and storage of a portion of it as the inert phosphate in the bone. We would expect the symptoms to subside when the concentration of lead in the soft tissues reached the range of "normal" lead, providing there had been no deterioration of tissue beyond the ability of the body to readily repair or regenerate it. Under these conditions, if storage of lead in the bone has not been too great, normal health may return with or without medical treatment, providing the person has been removed long enough from the lead exposure. Abnormal fluctuations in the lead content of the blood and urine will be observed for a considerable length of time until an equilibrium is established between the lead stored in the bone on one hand, and lead in the soft tissues and body fluids on the other. At this time, excretion keeps pace with absorption, accumulation of lead ceases, and symptoms subside. It is unreasonable to believe that in such a person an acidosis from an infectious disease or an alcoholic debauch would again result in an acute attack of lead poisoning for two reasons: first, the remaining lead stored in the bone is probably so compact, insoluble, and of such a low concentration that not enough is available to produce intoxication again; second, if such were true, most normal persons would be subject to acute attacks of lead poisoning during acidosis, or alkalosis. As proof of this, there was no evidence of lead intoxication during illness among any of the individuals in the "normal" or "unjustified" groups, who died as a result of some acute infectious disease. A recurrence of plumbism after removal from the lead exposure indicates that insufficient time (probably about two years) has elapsed for the body to reach its normal equilibrium by the gradual excretion of lead stored in it, providing there has been no permanent damage to certain tissues.

Not only those individuals who have never been exposed to abnormally high concentrations, but also those who have at some time been exposed slightly but developed no signs or symptoms of intoxication, shall be considered as normal subjects. The concentration of lead in the respective tissues and fluids of this group will be designated as "normal" lead.

The toxicologist is often called upon to testify as to the results of his analysis. He may be asked to give his opinion as to whether or not the lead content of the specimens was normal or above normal. It is for this reason that the concentration of lead was determined in the tissues of 25 individuals who were never known to have exhibited any signs or symptoms of lead intoxication.

Group A. Four Fetuses and One Nursing Infant

THE five cases in this group include two stillbirths who never breathed. In other words, there was no chance of lead contamination from any external source such as milk or air, but traces of lead were found in the tissues. The mothers gave no histories of lead exposure other than that of the ordinary housewife, and never had any symptoms of lead intoxication. The same is true

TABLE III

"NORMAL" LEAD CONTENT OF TISSUES IN FOUR FETUSES AND ONE NURSING INFANT
CONCENTRATION (mgm. Pb per 100 gm.)

Cases No.	Age	Cause of Death	Liver	Kidney	Bone (Sternum and Ribs)
1	2 hours	Birth injury	0.011	0.028	0.110
2	Born dead	Still-birth	0.049	0.021	0.090
3	Born dead	Still-birth	0.047	0.019	0.050
4	1 hour	6 mo. prematurity, congenital atelectasis of pulmonary lobes	0.030	0.014	0.006
5	4 months	Internal hydrocephalus	0.045	0.024	0.005
Mean			0.036	0.021	0.052
Probable error of mean			±0.007	±0.002	±0.026

of the mothers of the other three. In Cases 1 and 4 there was breathing for two hours and one hour respectively, but neither had nursed. Case 5 is that of an infant who nursed at the breast for four months.

The summary of these results will be found in Table III.

The concentrations are only a fraction of those in the older normal groups but they show that lead is present in the earliest stages of life. A possible explanation of the lower values in the fetus as compared to those of the mother may lie in the fact that only a very small percentage of the mother's blood reaches the uterus. Much of the lead could be lost during the process of osmosis and infiltration of blood and other fluid across the uterine and fetal villi before passing down the umbilical vein to the fetus.

The specimens were handled very carefully. They were placed in lead-free containers, and instead of mincing in a metallic grinder, were sliced with a fine steel knife on a board, both of which were cleaned meticulously. We may therefore conclude that the viscera of the fetus contain traces of lead, apparently harmless, and undoubtedly coming from the blood and tissue fluids of the mother. The fact that the fetal tissues showed a much lower concentration of lead than those of mothers and housewives (see Tables V, VI, and VII) is evidence against the selective absorption of lead by the fetus. The increase observed in older children (Table IV) is undoubtedly due to food and drink, and to lead encountered in ordinary childhood play. Prematurities and miscarriages often blamed on lead are most likely due to the effect of lead on smooth muscle (the uterus). Infants of leaded mothers have died in convulsions or suffered permanent impairment as found by Sir Thomas Oliver[41] and others, but so far as is known the concentration of lead in the fetal tissues has not been demonstrated to be higher than in those of the mother. However, much more iron and copper was observed in the fetal livers than in those of older persons.

Tompsett and Anderson[21] found from two to three times as much lead in the tissues of four fetuses which they examined, as compared to those reported here. This may be due to a geographical difference.

Kehoe[40] found small amounts of lead in some tissues and none in others of five fetuses, possibly explained by the fact that at that time the more sensitive and accurate dithizone method was not available.

TABLE IV

"NORMAL" LEAD CONTENT OF TISSUES IN FIVE CHILDREN
CONCENTRATION (mgm. Pb per 100 gm.)

Case No.	Age (yrs.)	Cause of Death	Liver	Kidney	Bone*
6	5	Struck by automobile, causing traumatic rupture of liver	0.228	0.085	1.015
7	2	Tuberculous leptomeningitis	0.297	0.073	1.488
8	12	Tuberculosis and generalized amyloidosis	0.059	0.038	0.524
9	11	Died following mastoidectomy	0.277	0.143	3.112
10	1¼	Diabetes mellitus and acidosis	0.050	0.110	
Mean			0.182	0.090	1.535
Probable error of mean			±0.053	±0.018	±0.561

*Sternum and ribs, mostly cartilage.

TABLE V

"NORMAL" LEAD CONTENT OF TISSUES IN FIVE YOUTHS

CONCENTRATION (mgm. Pb per 100 gm.)

Case No.	Age (yrs.)	Occupation	History and Clinical Findings	Postmortem Findings	Cause of Death	Liver	Kidney	Bone
11	21	Oiler for 6 months at harvester company	None	Multiple gun-shot wounds	Gun-shot wounds of head and trunk, died suddenly	0.218	0.168	1.200
12	19	Unemployed	Pains in abdomen, vomited clots of blood after whiskey bouts	Diapedesis, alcoholic cirrhosis of liver	Cirrhosis of liver	0.100	0.069	0.925
13	19	Housework at home	Very obese, epilepsy, profuse vaginal hemorrhage	Enlarged pituitary weighing 13 grains	Profuse vaginal hemorrhage	0.098	0.042	0.730
14	20	Unemployed	Took Lysol in store, died few hours later	Erosion of G.I. tract pregnant uterus, 6 mo.	Acute Lysol (cresol) poisoning	0.074	0.045[a]	0.448
15	24	Unemployed	Chronic alcoholism, alcoholic psychosis or avitaminosis	Bronchopneumonia, chronic myocarditis, pseudo-pellagra.	Chronic myocarditis and bronchopneumonia	0.237	0.071[b]	2.032
	Mean					0.145	0.079	1.067
	Probable error of mean					±0.034	±0.023	±0.271

a—0.032 in medulla; 0.047 in cortex
b—0.064 in medulla; 0.074 in cortex

Group B. Five Children

THE results are summarized in Table IV. The children varied in age from one year and three months to 12 years. The concentration of lead in the liver and kidneys was approximately five times greater, and in the bone, about 29 times more than those of Group A, Table III. In spite of this fact, no signs or symptoms of lead poisoning were observed and there was no history of an abnormal exposure to lead. It is of interest to observe the increase of lead. In Group A the only exposure was internal, the blood of the mother, and, in one case breast milk for a short time. In Group B the exposure was external, but such factors as lead-painted furniture and lead toys, as far as could be determined, were ruled out. Children in this age

TABLE VI

"NORMAL" LEAD CONTENT OF TISSUES IN FIVE MIDDLE-AGED INDIVIDUALS

CONCENTRATION (mgm. Pb per 100 gm.)

Case No.	Age (yrs.)	Occupation	History and Clinical Findings	Postmortem Findings	Cause of Death	Liver	Kidney	Bone
16	36	Housewife	Suspected gastric ulcer, received bismuth subnitrate, coughed up bloody, frothy material	Decompensated heart, edematous, luetic	Decompensated heart	0.223[a]	0.115[a]	1.240[a]
17	39	Housewife	Very obese, fainted and became unconscious at home	Cerebral hemorrhage	Cerebral hemorrhage	0.262	0.090	1.248
18	32	Domestic servant	Poorly nourished, moderate drinker	Abdominal stab-wound, peritonitis	Peritonitis following stab-wound in abdomen	0.233	0.111	0.740
19	45	Waiter	Comatose for three days, heavy drinker	Skull fracture	Skull fracture	0.307	0.112	1.528
20	34	Cook	Seized with epigastric pain, vomited blood after eating candy, jaundiced for years	Rupture of varix of esophagus	Hemorrhage from rupture of varix of esophagus	0.112	0.054	0.598
	Mean					0.227	0.096	1.071
	Probable error of mean					±0.032	±0.017	±0.187

a—Bismuth eliminated as described in "The Photometric Dithizone Method."

group will crawl on the floor or ground and put their hands into their mouths. They like to play in dirt in yards, on playgrounds, and in the streets where the exhaust material from automobiles possibly increases the exposure to lead. These conditions are not abnormal. They are what we normally expect.

The concentration of lead in this group has increased to the point where it is approximately the same as will be found in the older-age groups. Because no signs or symptoms of intoxication were observed, this concentration may be justly called "normal" lead.

Group C. Five Youths

THE subjects varied in age from 19 to 24 years. The average concentrations of lead are slightly less but of the same order as those of Group B, Table IV. The results are summarized in Table V.

The added exposures here as compared to Group B were of the nature of normal external exposures of longer duration, but there was no evidence of an abnormal exposure or lead intoxication.

At this time the thought occurred that the medulla or tubular portion of the kidney might contain more lead than the cortex, or vice versa. In Cases 14 and 15, three determinations were made on each kidney: one on cross sections through the whole kidney, another on the medulla, which was stripped as cleanly as possible from all parenchymatous tissue with a fine pointed sharp knife, and a third on the parenchymatous tissue of the cortical area. The results showed that the medulla contained a slightly smaller amount of lead than the cortex.

Group D. Five Middle-Aged Individuals

THE ages varied from 32 to 45 years. The average concentration of lead was quite similar to that of the two previous groups with the exception that it was slightly higher in the liver. The results are found in Table VI.

Group E. Five Aged Individuals

THE ages varied from 50 to 84 years. Again the lead content of the tissues is of about the same order as that of the three previous groups. The liver and kidney showed slightly less lead and the bone slightly more than those of the youths in Group C and the middle-aged Group D.

Case 25 was accepted as normal because the chemical analysis revealed a lead concentration well within the normal range and there were no signs or symptoms of plumbism, in spite of the fact that this man had been a brush painter for years. This can be explained in two ways, either the paint contained no lead pigment, or the necessary precautions were used to avoid the absorption of toxic quantities. The same explanation would apply to the unverified cases 38, 39, 43, and 45 of painters in Table XII. Another consideration is that these individuals may have developed symptoms at one time, but had been removed from the lead exposure sufficiently long to allow the lead concentration to drop to "normal."

The concentration of lead in this group is further evidence that the chemist may predict with a reasonable degree of certainty that symptoms of plumbism are usually absent when lead comes within this range.

The average concentration of lead in the tissues of 20 human beings (excluding fetuses in Table III) who died of various causes with no signs or symptoms of lead poisoning was as follows:

0.165 (probable error ±0.037) mgm. in the liver, varying from 0.038 to 0.307;

0.083 (probable error ±0.018) mgm. in the kidney, varying from 0.029 to 0.168;

1.258 (probable error ±0.323) mgm. in bone (sternum and ribs), varying from 0.398 to 3.112.

Calculations in the normal groups were carried

TABLE VII

"NORMAL" LEAD CONTENT OF TISSUES IN FIVE AGED INDIVIDUALS

CONCENTRATION (mgm. Pb per 100 gm.)

Case No.	Age (yrs.)	Occupation	History and Clinical Findings	Postmortem Findings	Cause of Death	Liver	Kidney	Bone
21	50	Housewife	Chronic alcoholic, body lice and very unclean	Chronic myocarditis	Chronic myocarditis	0.208	0.108	1.680
22	53	Street laborer	Chronic alcoholic, delirium tremens, pneumonia	Pneumonia	Pneumonia	0.103	0.068	2.025
23	59	Salesman ten years, retired 5 years	Old heart case, died in hospital examining room	Organic heart disease	Organic heart disease	0.075	0.075	1.340
24	84	Piano player and tuner	In hospital two days for carcinoma of stomach	Coronary thrombosis, arteriosclerosis, carcinoma of stomach	Coronary Thrombosis	0.038	0.029	0.398
25	60	Painting with brush and general labor	No lead signs or symptoms, died suddenly	Occlusions of coronaries, arteriosclerosis	Coronary occlusions	0.077	0.042	1.308
	Mean					0.100	0.064	1.350
	Probable error of mean					±0.029	±0.014	±0.273

out to the third significant figure to show a wider spread in the lower concentrations. In an actual case of lead poisoning, this figure is of no significance.

The "normal" lead concentration in human tissues has been determined by several workers. Meillère[42] found from 0.1 to 0.2 mgm. in the liver and spleen; Aub et al,[19] almost exclusively in the skeleton; Sheldon and Ramage,[43] occurring in some normal organs and none in others; Boyd and De,[44] in all organs except the brain; Weyrauch and Muller,[45] no appreciable amount in liver, kidney, spleen and brain. Lynch, Slater, and Osler[46] found 0.15 mgm. in some livers and kidneys and none in others. They found a concentration in the bone ranging from 1.45 to 14.6 mgm., considerably higher than those in the present paper, but explainable by the fact that they worked on shafts of femur and tibia (known to have higher concentrations, as shown by Tompsett[20] and Aub[19]), whereas sternum and ribs only were available for the present studies. In two cases Kehoe[40] found amounts in the corresponding tissues comparable with those reported here, but negative results in other tissues. Tompsett and Anderson[21] examined 20 cases and found lead in all tissues. The size of their series and the concentrations (0.173 in the liver, 0.135 in the kidney, and 0.855 in the rib) are in close agreement with those of this paper.

The following conclusions may be drawn:

1. That human beings are born with a very small concentration of lead in the various tissues, averaging about 0.04 in the liver 0.02 in the kidney, and 0.05 of a milligram in the cartilaginous material. The lead is undoubtedly derived from the blood and body fluids of the mother, but is of a lower concentration than that of corresponding maternal tissues.

2. That from the pre-school age until very old age the lead reaches an average concentration of approximately 0.2 in the liver, 0.1 in the kidney, and 1.3 milligrams in the bone. This concentration is derived from slight exposures at work and play, and from food, drink, and the atmosphere.

3. That the concentration reached during the pre-school age in the respective tissues seems to remain at a fairly constant level all through life, in the absence of excessive exposure. The body appears to establish a state of equilibrium to combat the daily intake of small non-toxic quantities of lead. The greater part is eliminated without being absorbed. Some of the absorbed lead is stored in the bone as the inert phosphate, most is eliminated through the urine and feces, and a very small part is found in the viscera and circulating blood. The storage of lead in the bone must be limited by conditions of normal equilibrium, otherwise the constant intake of small non-toxic amounts would build up the concentration to the point where an acidosis would develop acute symptoms of lead poisoning. Such has not been the case in the experience of the writer. It is only

TABLE VIII

"NORMAL" CONCENTRATION OF LEAD IN URINE

Case No.	Occupation	Abnormal Lead Exposure	Mgm. Pb 1000 cc. Urine
1	Chemist	None	0.007
2	Chemist	None	0.011
3	Chemist	None	0.018
4	Toxicologist	None	0.015
5	Serologist	None	0.005
6	Physician	None	0.021
7	Physician	None	0.032
8	Physician	None	0.027
9	Physician	None	0.019
10	Clerk	None	0.022
11	Stenographer	None	0.017
12	Unemployed 2 years	Brass Molder	0.059
13	Unemployed 6 months	Occasional painting	0.078
14	Unemployed 2 years	Painted stoves black	0.020
15	Unemployed 6 months	Painted stoves	0.053
16	Unemployed 3 months	Painter	0.065
17	Unemployed 4 months	Lighting solder pots	0.007
18	Unemployed	Painter	0.064
Mean			0.030
Probable Error of Mean			±0.0048

true where the exposure and concentration is considerably above the normal.

4. That the daily intake of small non-toxic amounts of lead does not cause an ever-increasing concentration in the tissues and a cumulative effect. Instead of this, lead accumulates to a certain degree, any further slight excess is thrown off, and no effects are observed. However, it does seem that of all the common metallic poisons, lead is the most cumulative. If the exposure is excessive, the equilibrium of the body is disturbed, absorption and storage steadily increase the accumulation of lead which in turn may cause a cumulative effect on certain tissues to a point beyond repair.

5. That the concentration of lead constantly found in tissues of individuals who have no signs or symptoms of lead poisoning, and who have not been exposed to excessive or toxic amounts, may be called "normal" lead.

The "Normal" Concentration of Lead in Body Fluids

URINE—The group of normal subjects may be divided into two sub-groups: (a) those having no lead exposure other than that which occurs under ordinary living and working conditions (Cases 1 to 11), and, (b) men who had no symptoms but whose former occupations were such that slightly abnormal quantities of lead might have been absorbed (Cases 12 to 18).

Case 2 is that of the writer who never had an abnormal exposure or the slightest indication of lead intoxication.

Case 17 here is the same as that of Case 45, Table XII, in which lead poisoning was suspected because he lit solder pots in the morning. From the analysis of the urine, while the patient was still living, the writer offered the opinion that lead poisoning was not indicated. He died later and the anatomical findings showed that the immediate cause of death was uremia caused by congenital atrophy and malformation of both kidneys and ureters. The chemical analysis of the organs revealed that lead was well within the normal range, which fact was correlated with the urinary findings, and was substantial evidence against the possibility of lead intoxication.

The average concentration of lead in the urine of these 18 individuals was 0.03 mgm. This value is slightly less but of the same order of magnitude as that found by Kehoe[47] (0.04 mgm.). Lead within this range has been found in the urine of more than 100 patients admitted to the Cook County Hospital, who were suspected cases of plumbism, but unjustified in the light of subsequent findings.

BLOOD—It was hoped that the "normal" concentration of lead in the blood could be determined, but the idea was discarded due to lack of a sufficient number of volunteers. However, Kehoe[47] has reported this value as 0.06 mgm. per 100 cc., as determined spectrographically. Kaplan and McDonald[48] found the "normal" lead in blood to be 0.03 mgm. per 100 grams in 126 adults and children, which value seems to be the more acceptable.

Bibliography:

1. DUCKERING, G. E.: Methods of Determination of Dust and Lead in the Air of Workrooms. Report Chief Inspector of Factories, 201, 1910.

2. RUSSEL, A. E., et al.: Lead Poisoning in a Storage Battery Plant, *U. S. Pub. Health Bull.*, 205:55, 1933.

3. QUAN, G. N., and KLEIN, A.: Lead Pipes as a Source of Lead in Drinking Water. *Am. J. Pub. Health*, 26:778, 1936.

4. Deaths from Lead Poisoning, *U. S. Bur. Labor Statistics, Bull.* 426, 1927.

5. DUFOUR-LABASTIDE, A.: L'intoxication Saturnine chez L'enfant, *These de Paris*, 1902.

6. SUZUKI, T., and KANEKO, J.: Serous Meningitis in Infants Caused by Lead Poisoning from White Powders. *J. Orient. Med.*, 2:55, 1924. Abst. *J.A.M.A.*, 82:1819, 1924.

7. FUKUSHIMA, M., and MATSUMOTO, H.: Statistics of 298 Cases of Infantile Lead Poisoning, *Orient. J. Dis. Infants*, 3:27, (March) 1928.

8. LEWIN, L.: Das Toxische Verhalten von Metallischem Blei und besonders von Bleigeschossen in thierischen Korper. *Arch. f. Klin. Chir.*, 94:937, 1910-11.

9. CAMPANER: *Rev. espan. de Med. y Cir.*, 7:328, 1924 (Quoted in Legal Medicine and Toxicology, by Ralph W. Webster, 1930).

10. UTTAL, J.: Chronic Lead Poisoning from Snuff; Report of three cases. *J.A.M.A.*, 90:288, 1928.

11. BAUER, W., and ROPES, M. W.: Case of Lead Poisoning Resulting from Use of Snuff; Lead Studies, *Ibid.*, 757.

12. LEGGE, T. M., and GOADBY, K. N.: Lead Poisoning and Lead Absorption, 1912, London and New York.

13. MINOT, A. S.: Lead Studies: V. The Distribution of Lead in the Organism after Absorption by the Respiratory Tract and Subcutaneous Tissue. *J. Indust. Hyg.*, 6:137, 1924.

14. BLUMGART, H. L.: Lead Studies: VI. Absorption of Lead by the Upper Respiratory Passages. *J. Indust. Hyg.*, 5:153, 1923.

15. FAIRHALL, L. T.: The Solubility of Various Lead Compounds in Blood Serum. *J. Biol. Chem.*, 40:481, 1924.

16. FINE, J.: The Phagocytosis of Lead Compounds and Their Influence on the Activity of the Leukocyte. *J. Indust. Hyg.*, 5:138, 1923.

17. DRINKER, C. K.: Modern Views Upon the Development of Lung Fibrosis. *J. Indust. Hyg.*, 3:295, 1921-22.

18. CARLSON, A. J., and WOELFEL, A.: The Solubility of White Lead in Human Gastric Juice and Its Bearing on the Hygiene of the Lead Industries. *Am. J. Pub. Health*, 3:755, 1913.

19. AUB, J. C., FAIRHALL, L. T., MINOT, A. S., and REZNIKOFF, P.: Lead Poisoning, Medicine Monographs: VIII, Williams and Wilkins Company, Baltimore, 1926.

20. TOMPSETT, S. L.: Distribution of Lead in Human Bones. *Biochem. J.*, 30:345, 1936.

21. TOMPSETT, S. L., and ANDERSON, A. B.: The Lead Content of Human Tissues and Excreta. *Ibid.*, 29:1851, 1935.

22. KEHOE, R. A., and THAMANN, F.: The Behavior of Lead in the Animal Organism: III. Colloidal Lead Compounds. *J. Lab. & Clin. Med.*, 19:178, (November) 1933.

23. HUNTER, M., and AUB, J. C.: Lead Studies: XV. The Effect of the Parathyroid Hormone on the Excretion of Lead and of Calcium in Patients Suffering from Lead Poisoning. *Quart. J. Med.*, 20:123, 1926-27.

24. AUB, J. C., ROBB, G. P., and ROSSMEISL, E.: Significance of Bone Trabeculae in the Treatment of Lead Poisoning. *Am. J. Pub. Health*, 22:825, 1932.

25. FISCHER, H.: Detection of Heavy Metals with the Aid of Dithizone (diphenylthiocarbazone). *Angew. Chem.*, 42:1025, 1929.

26. FISCHER, H., and LEOPOLDI, G.: Microchemical Determination of Heavy Metals with Dithizone (diphenylthiocarbazone). *Wiss. Veröffentlich. Siemens-Konzern*, 12:44, 1933.

27. WICHMAN, H. J., MURRAY, C. W., HARRIS, M., CLIFFORD, P. A., LOUGHREY, J. H., and VORHES JR., F. A.: Methods for Determination of Lead in Foods. *J. Assoc. Off. Agr. Chem.*, 14:108, 1934.

28. FAIRHALL, L. T.: Lead Studies: I. The Estimation of Minute Amounts of Lead in Biological Material. *J.*

Indust. Hyg., 4:9, 1922; *J. Biol. Chem.*, 60:485, 1924.

29. Clifford, P. A., and Wichman, H. J.: Dithizone Methods for the Determination of Lead. *J. Assoc. Off. Agr. Chem.*, 19:130, (February) 1936.

30. Wilkins, E. S., Willoughby, C. E., Kraemer, E. O., and Smith, F. L.: Determination of Minute Amounts of Lead in Biological Materials. *Indust. & Eng. Chem., Anal. Ed.*, 7:33, 1935.

31. Horwitt, M. K., and Cowgill, G. R.: A Titrimetric Method for the Quantitative Estimation of Lead in Biological Materials. *J. Biol. Chem.*, 119:553, 1937.

32. Seelkopf, K., and Taeger, H.: Quantitative Bestimmung Kleiner Bleimengen, *Ztschr. f. d. ges. exper. Med.*, 91:539, 1933.

33. Winter, O. B., Robinson, H. M., Lamb, F. W., and Miller, E. J.: *Indust. & Eng. Chem., Anal. Ed.*, 7:265, 1935.

34. Ross, J. R., and Lucus, C. C.: A New Method for the Determination of Minute Amounts of Lead in Urine. *J. Biol. Chem.*, 3:285, 1935.

35. Willoughby, C. E., Wilkins, Jr., E. S., and Kraemer, E. O.: *Indust. & Eng. Chem., Anal. Ed.*, 7:285, 1935.

36. Hubbard, D. M.: Determination of Lead. A Photometric Dithizone Method as Applied to Certain Biological Materials. *Indust & Eng. Chem.*, 29, *Anal. Ed.*, 9:493, October 15, 1937.

37. Kehoe, R. A., Thamann, F., and Cholak, J.: On the Normal Absorption and Excretion of Lead: 1. Lead Absorption and Excretion in Primitive Life. *J. Indust. Hyg.*, 15:257, 1933.

38. Kehoe, R. A., Thamann, F., and Cholak, J.: On the Normal Absorption and Excretion of Lead: II. Lead Absorption and Lead Excretion in Modern American Life. *Ibid.*, 273.

39. Kehoe, R. A., Thamann, F., and Cholak, J.: On the Normal Absorption and Excretion of Lead: III. The Sources of Normal Lead Absorption. *Ibid.*, 290.

40. Kehoe, R. A., Thamann, F., and Cholak, J.: On the Normal Absorption and Excretion of Lead: IV. Lead Absorption and Excretion in Infants and Children. *Ibid.*, 301.

41. Oliver, Sir Thomas: Lead Poisoning, 1914, London and New York.

42. Meillere, G.: Le Saturnisme, etude historique physiologigue, clinique et prophylactique, Paris, (1903).

43. Sheldon, J. H., and Ramage, H.: Spectrographic Analysis of Human Tissues. *Biochem. J.*, 25:1608, 1931.

44. Boyd, T. C., and De, N. K.: Some Applications of Quantitative Spectroscopic Analysis. *Indian J. M. Research*, 21:109, July, 1933.

45. Weyrauch, F., and Muller, H.: Das Sogenannte normale Blei im menschlichen Körper. *Ztschr. f. Hyg. u. Infektionskr.*, 115:216, 1933.

46. Lynch, G. R., Slater, R. H., and Osler, T. G.: Determination of Traces of Lead in Biological Materials, with Special Reference to Bone. *Analyst*, 59:787, 1934.

47. Kehoe, R. A., Thamann, F., and Cholak, J.: Normal Absorption and Excretion of Lead. *J.A.M.A.*, 104:90, January 12, 1935.

48. Kaplan, E., and McDonald, J. M.: The Blood Lead Value as an Aid in the Diagnosis of Lead Poisoning. *The Scientific Proc. of Am. Soc. Pharmacol. & Exper. Therap.*, 29th Annual Meeting, March 30-April 12, 1938.

The Treatment of Burns

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THE treatment of burns is quite well standardized.* There are many variations of the general principles of the treatment, as set up by Davidson, with the use of tannic acid. There have likewise been several substitutes for the use of tannic acid in the first stages of burns, all of which, however, revert to the main principle of prevention of absorption of the toxic products and giving a protective covering to the burned area.

The first and primary principle in the treatment of burns is the prevention of shock. The best ways to prevent shock are to cover the burned area with a coagulating substance, of which I believe tannic acid is far superior to any in use to date, and to administer large doses of morphine. In my experience, the method of applying tannic acid jelly to the burned area and covering of the wound immediately with sterile gauze is superior to the tannic acid spray. I believe that exposure of a severely burned person to the air, which is essential in applying tannic acid by the spray method, exposes the patient to much greater shock than the application of the jelly and the immediate covering of the wound with sterile dressings. Extensive burns involving the hands are best treated with an ointment of the butesin picrate type. By using this type of ointment on the hands, one will avoid the embarrassing constriction of the eschar which always takes place when tannic acid is applied to extensive burns of the hands. If tannic acid has been applied, the tight constricting eschar must be incised by longitudinal incisions so as not to shut off the blood supply if the constriction becomes too tight.

In the presence of shock, hypertonic glucose solutions, together with morphine, should be administered. The fluid intake and output should be watched carefully so as not to allow the patient to become dehydrated or oversaturated. After 48 to 60 hours, burned extremities may be soaked in normal saline and movement of the fingers begun. During this stage, the red count must be watched carefully for a beginning anemia. A falling red count with an increase in toxemia should be treated by transfusion of whole blood.

After the application of moist saline dressings or soaks, an ointment composed of 50% cod liver oil mixed with a 10 or 20% balsam Peru is an excellent protective covering which keeps the eschar soft and does prevent infection. It should be remembered that boric acid and tannic acid are incompatible, and boric acid should at no time be used where tannic acid has previously been applied. I have had excellent results by the mere application of this ointment over the eschar and waiting for granulation to occur under the eschar. If, after 10 days to two weeks, the eschar does not loosen, it will have to be removed surgically. Cod liver oil ointment is applied to the denuded area until a suitable granulating surface occurs, upon which pinch grafts can be applied. The pinch grafts should be applied to the granulating surface regardless of whether or not infection is present. I have used the Reverdin type of graft in the presence of severe infection with excellent results. For the first 24 hours following application of the grafts, saline dressings were applied; after that the cod liver oil-balsam Peru mixture was applied daily. In order to prevent

* From a paper presented at the last annual clinical meeting of the Central States Society of Industrial Medicine and Surgery.

loosening and slipping of the grafts, Abbott's par-resined lace-mesh was applied over the graft and left intact for 10 days. Pressure dressings are applied over the mesh. This facilitates changing of dressings without danger of disturbing the grafts under the mesh. After 10 days, this dressing may be soaked off in normal saline and the hands exposed to the air daily. It has been my experience to have a 100% take of the Reverdin type of graft in the presence of infection and a four plus Wasserman when the cod liver oil-balsam Peru mixture was used. Occasionally when healing occurs, some very large annoying keloids will develop, which respond excellently to x-ray therapy if administered early and of sufficient dosage.

It may be added that severe burns which result from explosions very often have pulmonary involvement. This type of patient should be given the advantage of administration of oxygen by means of the nasal catheter method. I have had temperatures drop from the vicinity of 104° to normal within 12 hours following the administration of oxygen to this type of patient. Of course there is a secondary rise, but the improvement with oxygen is tremendous.

SUMMARY: In summarizing, it is obvious that the first principle in the treatment of burns should be the prevention of shock, which is best carried out by the immediate covering of the burned area with a preparation of tannic acid jelly. The use of cod liver oil-balsam Peru mixture must be had to be appreciated. Blood transfusions and intravenous glucose are to be used when indicated.

Skin grafting of the Reverdin type should always be done on large denuded areas. This should be done as soon as the granulations permit, regardless of whether or not infection is present. Oxygen should be administered to patients with burns resulting from explosion where there is pulmonary involvement. Keloids formed during healing should be treated by x-ray therapy early to obtain the best results.

Cancer

—Progress Report on the "APSA" Treatment—

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IN THE May, 1938, issue of INDUSTRIAL MEDICINE an article was published entitled: "Cancer—A New Treatment Holding Much Promise."* The substance used in this treatment has been given the name "APSA." The article contained a description of the technique of treatment by this method with a brief outline of the sequelae. A few case histories and photographs of patients before and after treatment were shown. The unusually large demand for reprints of this article has been very gratifying, since it tends to show the interest manifested by so large a group of the profession with open minds and non-skeptical views towards what *may possibly* prove to be a cure for that scourge of humanity, ranking only second to heart disease in the causation of death, and the long drawn-out suffering from which is scarcely comparable with that associated with any other malady.

The reaction of most physicians who have manifested an interest in this treatment has been generally favorable. Quite naturally some adverse criticism has arisen, but in each and every instance this has come either (1) from physicians who have not taken the time, trouble or pains to make an investigation but who are ever willing to condemn anything that does not emanate from themselves or in which they have not been taken into confidence; or (2) from those who think the formula should be given out at once. Regarding the first group, the writer has derived a considerable amount of interest from the various statements of those who are entirely in ignorance with reference to the matter and who do not appear to be desirous of enlightenment. This particularly in view of the fact that many cases previously treated by some of the critics—with failure to produce any benefit, and in some instances final admission that they could do no more for the patient—have come under the writer's observation and treatment with results that have been almost astounding.

In answer to the critcism that has arisen due to non-publication of the "APSA" formula, attention is called to the former article in which the effort was made to point out in a most lucid manner the fact that this treatment was entirely in an experimental stage and that time alone would prove its ultimate effectiveness. The profession has established the five-year period during which cancer cases must remain free from recurrences before they may be considered as cured. While many cases treated by "APSA" present every appearance indicating that the malignancy has been completely eradicated, the standards set by the profession must be adhered to and will not justify classing them as cured until they have survived the established period without recrudescence. The essential ingredient of "APSA," being of vegetable origin, can only be extracted from the plant at the proper stage of development. To publicize the formula at the present moment would be an injustice to the profession, to the patient, and to the author. The method of producing "APSA" is quite intricate, and deleterious changes will follow the very slightest deviation from the process. Should persons unqualified for the production of this product attempt to produce it, or those untrained in the technique of its use attempt its application, disastrous effects might ensue—casting opprobrium upon an apparently valuable remedy. Just as soon as the experimental stage has sufficiently progressed to remove doubt from the mind of the profession and establish its real merit, the formula will be given to the profession and "APSA" will be made available through the medium of a reputable pharmaceutical house.

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* Vol. 7, No. 5, pp. 265-271.

Since the first announcement of "APSA," many cases have been treated with what appear to be entirely satisfactory results. There is no panacea for cancer, but when between 80 and 85% of the cases treated respond with results that give every appearance of being cures, we are constrained to admit that such results are far in excess of those obtained by the formerly recognized and established methods of treatment—radium, x-ray and surgery—which are admittedly beneficial to only a limited degree and will affect normal as well as neo-plastic tissues. The advantages claimed for "APSA" are its peculiar selectivity for malignant tissues only, and the fact that its action ceases when the new growth has been destroyed and normal tissues are reached.

ruary 11, 1938, shows the mummified sequestrum separating from the normal tissue; this was lifted out, leaving the denuded area of normal granulating tissues—shown in the third picture. A mild ointment was applied daily to the granulating wound which, on March 19, 1938, had healed with the exception of a small area, as shown in picture No. 4. On April 23, 1938, the patient was seen, and the wound had completely healed, as shown in picture No. 5. Glands in the axilla could not be palpated, and the general appearance of the patient was greatly improved. Up to the present there had been no recurrence and the patient continues to be happy and apparently well.

A MOST interesting breast case is that of Mrs. W., age 56, whose left breast was amputated for

Fig. 1. Pictures 1, 2, 3, 4, 5

A case illustrative of the progress and action of "APSA" is shown in the series of pictures constituting Fig. 1. Mrs. C., age 71, first seen December 7, 1937, gave the history of having developed a growth in the left breast about two and one-half years previous. Amputation was advised but declined, and the lesion broke through the skin about April, 1937. X-ray and radium treatment were administered without benefit. To the lesion, approximately 3 inches by 2½ inches in size, mercurochrome had been applied daily for some time, accounting for the exceedingly dark appearance of the involved area in the first picture. Pathological report from the biopsy specimen shows the tumor to contain surface epithelium which was thin, and underneath which was found dense connected tissue containing nests and strands of epithelial cells which were round, hyperchromatic, contained niceoli and many mitotic figures; about the nests of epithelium was a marked infiltration of round cells. The diagnosis was: "Grade 3. Adeno-carcinoma of the breast." The lesion, as shown in the first picture, was denuded by a light surface curettage and "APSA" applied. The selectivity of "APSA" for malignant tissue was well demonstrated in this case in that as mummification advanced the long axis of the lesion changed so as to point towards the nipple, and as the destruction of the carcinoma progressed it reached quite well into the areola and almost to the nipple itself. The second picture, taken Feb-

carcinoma a little more than three years ago. The malignancy recurred a few months later and an attempt was made to excise same, without avail. X-ray and radium treatment were then administered without apparent benefit, as the malignant area increased both in size and depth. At the request of Mrs. W.'s physician, the writer first saw this patient at St. Joseph's Infirmary on May 26, 1938, (Fig. 2-A). The diseased area was irregularly circular in outline and approximately 6 inches in diameter. In depth, it extended entirely through the muscles, fascia and intercostal tissues down to the plura. The second rib was involved in what appeared to be a secondary infection; the third rib, also similarly involved, had completely lost its costal cartilage and a portion of the anterior extremity of the rib—about 3 inches in all; the fourth rib was likewise involved along its superior anterior border and the costal cartilage. In the bottom of the wound the plura was readily visible, while at the lower inner angle a slight portion of the pericardium was visible. The lung had not collapsed but could readly be seen through the plura lying on contact therewith. It was quite doubtful whether "APSA" would prove of any benefit to this patient, also certain dangers were involved in that if applied "APSA" would be expected to seek out the malignancy regardless of what tissues might be involved, or where the plura itself might be affected and caused to mummify and slough out. After consultation with her

physician, who explained the dangers to the patient and to her family, it was decided that, the case being moribund, and no other line of treatment offering any hope, no injustice would be done by proceeding with the treatment. Accordingly, on May 26, 1938, the incrustations were removed and "APSA" applied quite extensively to the involved area. The patient remained in the hospital until May 28—when she was permitted to go to her home and since that time has come to the office once a week for dressings and observation. Other dressings, merely keeping the wound clean as possible, were done, as required, at her home. On July 2 the picture (Fig. 2-B) was made. It shows distinctly the more or less mummified necrotic tissue separating from the surrounding tissues where the line of demarcation had originally formed. Biopsy was taken from the edges of the surrounding tissue and reported as negative. Three weeks later (July 23, 1938) the necrotic tissue had sloughed away, leaving what gives every appearance of being a non-malignant wound (Fig. 2-C), which has since begun to throw out granulations around the entire periphery as well as from the base. Several physicians, including her own, having recently seen this case, expressed the opinion that all malignancy has been removed from the wound. Just as soon as the diseased ribs sequestrate and can be removed a plastic graft will be made to cover the large defect that exists. The patient's health has improved and while it is possible, even probable, that some metastasis exists which may become manifest at any time, the area of malignancy which existed on May 26, 1938, has, as far as is it humanly possible to determine, been removed.

MRS. H., age 61, married, had her left breast amputated 20 years ago on account of malignancy. At the same time a hysterectomy was performed. Laboratory reports if made at that time are not now available. This patient was first seen August 1, 1938, at which time there were several enlarged papillae on the right posterior edge of the tongue, together with a small ulcerated fissure. The biopsy report of this case was: "A precancerous lesion, although malignant transformation has not yet taken place." "APSA" was applied with the result that an area approximately ½ inch by ⅜ inch became mummified and separated from the tongue. The area granulated rapidly, and now (September 6, 1938) appears to be completely healed. The location of this lesion would not permit making pictures thereof.

MR. M., age 67, married, presented himself for treatment April 30, 1938, at which time he was suffering from a malignant growth involving the tissues about the left eye in the region of the inner canthus. This condition started about 18 months previously. Biopsy report: "Epidermoid carcinoma. Grade I." "APSA" was applied to the lesion—proving the statement made in the former article to the effect that even if applied to the conjunctiva of a normal eye "APSA" was not irritant thereto. Fig. 3-A shows the patient as first seen. On the third day after treatment this man returned to his home and did not report back in Atlanta until 18 days later, when all evidence of disease had disappeared, as shown in Fig. 3-B (May 19, 1938). This man was exhibited before the Fulton County Medical Society at its May 19

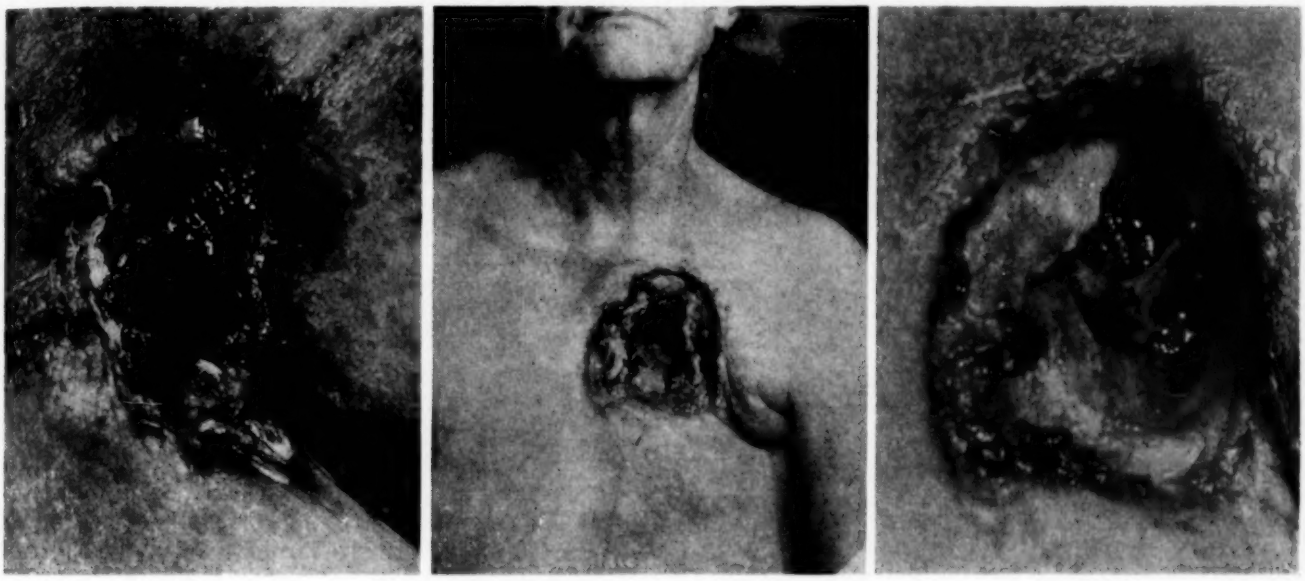

Fig. 2. A-B-C

Fig. 3. A-B

meeting and many of the physicians present examined him, all agreeing that the eye then presented an absolutely normal appearance.

ANOTHER case somewhat similar to the foregoing, except that it had been permitted to run for 12 years before applying for treatment, was that of Mr. L., shown in Fig. 4-A (June 1, 1938). In this case the malignancy had already destroyed the inner canthus of the left eye, the inner portion of both lids and the surrounding tissues; the conjunctiva and cornea had also become involved. Radium had been applied without avail. One application of "APSA" was made, and on August 13, 1938, all malignancy had apparently been eradicated. Naturally, the vision of this eye is impaired on account of the scar tissue involving the cornea. Fig. 4-B (September 10, 1938) shows the case as it is to date with the defect—which may easily be corrected by plastic surgery.

Fig. 4. A-B

ON June 29, 1938, Mr. McG., age 65, reported with an involvement of the right ear which had existed for about five years, and had been treated on several occasions with radium. The cartilage had been destroyed in part and the ear completely penetrated, as shown in Figs. 5-A (anterior view) and 5-B (posterior view). Biopsy taken was reported to be: "An epidermoid carcinoma. Grade 2."

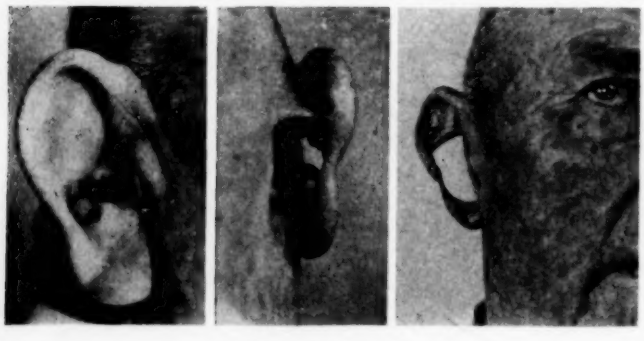

Fig. 5. A-B-C

One application of "APSA" was made, and on September 4, 1938, the malignancy had apparently been eradicated and the case ready for the plastic surgeon. The amount of tissue eradicated by "APSA" is shown in Fig. 5-C.

MR. F., age 42, had a black mole on the left side of his face. This broke down about two years ago, when a biopsy showed a melanoma of the mixed type. This was excised down to and including a lamina of bone and later a skin graft was made to cover the defect. The upper part of this graft remained healthy, but the lower portion broke down and another biopsy on July 13, 1938, revealed a "melanoma of the alveola type." Again, this was excised, but the patient became dissatisfied and first reported to the writer on July 28, 1938, the appearance of the lesion being as is shown in Fig. 6-A. "APSA" was applied, and Fig. 6-B shows the condition on August 5, 1938, when the line of demarcation had formed and the mummified malignant tissue may be seen separating from the normal surrounding tissues.

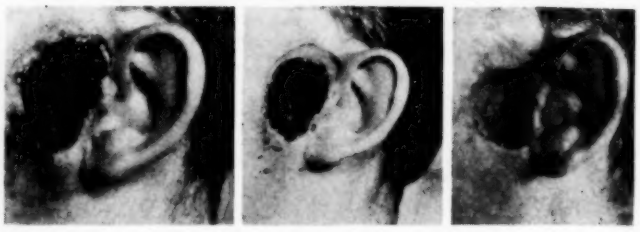

Fig. 6. A-B-C

Since August 5, the necrotic tissue has been cast off, leaving a granulating area, as shown in Fig. 6-C, which will shortly be ready for a skin graft.

MR. C. B., age 64, traveling salesman, was first seen July 15, 1938, when he gave the history of having a pimple develop on tip of nose about eight years previously. He received radium treatment but was left in condition as shown in Fig. 7-A. The entire tip of the nose and portion of the alae were involved; some loss of tissue had already taken place on the right side. The incrustation was removed and "APSA" applied; on August 12 the mummified mass came away, leaving granulating edges which have since healed. Fig. 7-B shows the condition at this time, the wound being thoroughly healed and ready for plastic surgery. The diagnosis in this case was "epithelioma."

To attempt description of all the cases treated would require an entire book. Many superficial cases were not photographed as their size or location precluded obtaining detail in pictures; these included epithelioma, papilloma, small basal cell carcinoma, sarcomona, etc., involving various parts of the face, the lips, and other portions of the body.

Fig. 7. A-B-C

MR. J. H. B., age 73, was suffering from a carcinoma of the esophagus located just a little below the level of the larynx. Obstruction was complete, a gastrostomy having been performed to admit of feeding. This man was first seen June

27, 1938 when, with the assistance of a competent bronchoscopist, a tube was passed and a tampon with "APSA" applied to the malignancy.

After a short period of time the patient insisted upon removal of the tampon, and left the hospital. He returned July 28, 1938, requesting another treatment, which was permitted to remain in place until the night of July 31, when it was removed, the patient leaving the hospital the following day. While this patent has not been seen for a little more than a month, it is reported that he can now swallow liquids in a normal way, is much improved in health, and contemplates returning for another treatment within the next few weeks. It is felt that this man has been greatly benefited and the case is reported merely as one of progress. We hope ultimately to restore this man's normal deglutition when the tube can be removed and the gastrostomy opening closed. Quite naturally pictures of such cases cannot be obtained.

THE first announcement of "APSA" (May, 1938) seemed to leave the general impression that only superficial cases were amenable to this therapy. Any lesion that may be reached through the natural body orifices and many that may be made accessible by, or in conjunction with, surgical procedures may be made suitable for the application of "APSA." These include lesions within the mouth, in the larynx, esophagus, rectum and cervix uteri.

Attention has been called to the fact that considerable edema and pain follow application of "APSA." Sight must not be lost of this edema in treating lesions in the throat and larynx; in all such cases laryngotomy should precede the treatment.

Prior to treating esophageal cases a gastrostomy is performed for the dual purpose of nourishing the patient and rendering it possible to maintain tampons in situ. Malignancies in the lower intestinal tract are only to be treated following colostomy; when all passages have been diverted from the lower bowel, application of "APSA" is made by means of tampons inserted through the proctoscope.

Malignant lesions of the vagina and cervix are readily accessible through the spectulum, responding quite readily to treatment.

Breast cancers that have not made their way to the surface are treated through crucial incisions into the tumor—when the biopsy has shown same to be malignant. The cases then follow the same routine as the one first illustrated in this article.

Space will not permit reporting all cases receiving "APSA" treatment in a single article. Mention of internal cases has been made above merely to correct the impression that "APSA" can only be applied in case of superficial malignancy. From time to time reports of special cases will be published, and, as far as practicable, will be accompanied by photographic illustrations.

The results obtained at our hands have been so encouraging, the number of cases treated exhibiting such a large percentage of apparent cures, we are constrained to feel that this product opens up a heretofore unknown therapy for malignancy, holding promise of great superiority over the present modes of treatment. It is earnestly hoped that further investigation and experimentation will lead to the development of an intravenous product from the active ingredient of "APSA," making practically all cancers that have not progressed beyond the bounds of hope amenable to this treatment.

Bernardino Ramazzini
(*1633-1714*)

—Founder of Industrial Medicine—

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OF ALL the Italian geniuses, such as Fallopius, Eustachius, Valsalva, Malpighi, Baglivi and Morgagni, Bernardino Ramazzini seems to stand alone, and alone to have fallen into oblivion. This great physician who was also a philosopher, physicist, poet and teacher was far in advance of his contemporaries of three centuries ago. His very radicalism, or innovations, might have been responsible for his historical eclipse. In the preface of his first work on industrial medicine he wrote: "I choose to publish this treatise of mine for the good of the Republic, or at least for the benefit of Tradesmen."

Ramazzini was born at Carpi on November 3, 1633, the son of Bartolomeo Ramazzini and Caterina Federzonia. He died at Padua on November 15, 1714. His early education was under Jesuit tutors. At the age of 19 he was sent to Parma to study philosophy, after which he matriculated in the school of medicine of that University and graduated on February 21, 1659. In Rome where he interned in the Metropolitan Hospitals he worked under Anton Maria Rossi. Upon leaving Rome he practiced in the jurisdiction of Canino and Marta, of the old Duchy of Castro of the Farnese Dynasty, but he was forced to relinquish this post, following a serious infection from malaria. He returned to his native town where he married Francesca Righi. From this union three children were born.

Strongly urged to settle at Modena in 1671, he finally decided to practice there. In 1682 he was elected full-fledged professor of medicine at the University of Modena, which had been greatly improved by Duke Francis II of Este. In this institution he remained 18 years teaching and practicing medicine, without neglecting the liberal arts for which he had a marked inclination. He was myriad-minded and embraced many different scientific fields, in each of which he became famous.

He initiated geologic researches by methods never attempted before, and inaugurated studies on variations of soil temperatures. The purity of water from its source to its distribution occupied

his scientific attention. The observations and interpretations of Giovanni Alfonso Borelli pertaining to barometric oscillations were rectified. He perceived a constant relationship between the barometric variations and the conditions of health, forerunning, therefore, the so-called "meteoropathology," and continued the observations concerning the distribution and diffusion of the diseases of man and animals.

He improved upon the concepts of Fracastorius on the various mechanisms of the propagation of contagion, apportioning a large part to the consideration of causes incident to environment, and increasing the value of deductions by statistical data.

He strenuously defended the use of quinine in the treatment of malaria and, according to Arturo Castiglione, was the first teacher of hygiene, studying man in relation to environment and establishing the deleterious action of the latter on the former, deducing the precautions to avoid or attenuate the consequences.

In 1700 he accepted the invitation of the Venetian Republic to fill the chair of medicine in the University of Padua. Notwithstanding his poor health, which forced him from time to time to suspend teaching, he dedicated himself to this with a great passion; even after becoming almost blind, he never interrupted his studies. The high esteem of the Senate procured for him a promotion.

He corresponded freely with the major part of learned men of his age, and belonged to numerous scientific societies.

THE chief scientific work of Ramazzini was accomplished in physics and meteorology, particularly as related to medicine. In epidemiology and clinical medicine he was the creator of the systematic study of industrial diseases and of the social defense of the workmen with whom he chatted and tried to know their work.

He studied the causes of absenteeism. On the basis of his original investigations he classified the various industrial diseases. He listed the diseases caused by the materials wherewith they came in contact, as the first division of industrial diseases. He next listed the diseases due to posture while on the job, as the second division.

With the keen insight of a philosopher he remarked: "It seems to be a doubtful case, whether we ought to reckon it good and pious office to prescribe physic for such wretches and thereby prolong a miserable life." But he added significantly: "Great profit arises to princes and merchants from mines and the use of metals is highly necessary to almost all arts."

He made health surveys of distant industrial centers and obtained from learned friends abroad reports on the state of health of workmen and gave them credit for such information. He read all the works of his contemporaries.

Ramazzini investigated and recorded his findings carefully. He studied each and every industrial process, and the diseases arising out of and in the course of such employment. He studied those engaged as metal diggers, workers in gilds, physicians, and others employed in mercury rubbing.

He refers to Fallopius "that Jacobius Carpenis received fifty thousand ducats of gold, solely by curing the pox by inunction; and that he killed many, though he cured by far the greater part. "It is certain," he said, "that this anointer knew better than the alchemists how to make a true metamorphosis of mercury into gold."

He studied chemists, potters, workers in copper and tin, glass makers, glass grinders and painters. He remarked that the painter, who sucked his brush tainted his stomach and intestines: lead colic. In the chapter on brimstone workers he criticizes practitioners who do not differentiate the different types of sulphur used in pharmacy. This same criticism holds today.

Ramazinni studied tanners, cloth cleansers, oilmen, blacksmiths, plaster of Paris, lime workers, cheese makers, and apothecaries. In all these occupations he advocated cleanliness as a preventive of skin affections. He commented on the morticians of his day, on the hygiene of midwives and nurses. He revealed his knowledge of the transmission of syphilis from the nurse to the infant and vice versa.

He next focused his attention on stone cutters, masons, bricklayers, brewers, bakers, millers, starch workers, washerwomen and laundresses.

Without the use of the microscope Ramazinni observed little worms in the corn which caused trouble to the sifters and meters of corn. With a prophetic eye this sage anticipated our present day knowledge on vitamines, ultra-violet rays and irradiation of food by the following paragraph:

"The Republic of Lucca have a wise custom of taking the corn out of their public granaries every year in the month of August and exposing it for some days to the sun beams."

Ramazzini described dermatitis from picking or hatchelling flax, hemp, or silk. None of the gamut of human activities escaped his keen observation.

He commented on guides and servants who attend baths, fishermen, and marines, and those engaged in salt pits. He described the disease of the Jews who led a sedentary life, and covered the couriers and wrestlers; music masters, singers and farmers came within his scope.

On the immunity of physicians from disease he gives this interesting account: "I have often wondered how it came to pass that when epidemic diseases, such as malignant fevers, pleurisy, and other popular sicknesses, were raging all about the practitioners of physic escaped free by a certain privilege as if it were tacked to their profession. And upon mature consideration I am of the opinion this good luck is owing to their caution, to their great exercise, and to the cheerfulness of mind when they return with full pockets from visiting their patients. In earnest, I have never observed the physicians to be so much out of order, as when nobody else is sick. And this I have seen verified in a particular manner these last five

years, which have been very healthy, without the appearance of any epidemic distemper. However, physicians do not always escape with impunity."

I wonder what would have been his comments on the present depression.

Ramazzini carried his studies further by the first description on the writers' cramp. He described the diseases associated with the occupation of weavers, carpenters, brass workers, brick makers, soap boilers, and sailors.

In the 17th century this great sanitarian was primarily absorbed in the health of the workmen and the prevention of occupational diseases.

He made epidemiological studies of disease; noted the atmospheric agents in the development of diseases. He invented the oblique mercurial barometer, and was a forerunner of atmospheric electricity and the discovery of oxygen and ozone.

IN HIS opus magnum, De Morbis Artificum, he advocated that civil legislation on behalf of the workmen should be accompanied by a medical program to preserve them from industrial diseases in the various types of activities.

His principal publications were:

Relazione sopra il parto e la morte della Ill. ma Signora Marchesa Martellini Bagnesi, ecc. Modena 1681; In solemni Mutinensis Academiae instauratione oratio, ecc., ivi 1683; De Fontibus Mutinensium admiranda scaturigine tratatus, ivi 1691; De Constitutione anni 1690 ac de rurali quae Mutinensis agri, ecc, ivi 1691; De Constitutione anni 1691 apud Mutinenses dissertatiokivi 1692; De Constitutione anni 1692, 1693, 1694, ivi 1695, Ephemerides barometricae ecc., ivi 1695 Dissertatio epistolaris circa mercurii motiones in barometro, ecc, ivi 1698; De Principum valetudine tuanda, ecc. Padova 1710; De Morbis artificum, ecc., Modena 1713 De contagiosa epidemiaquae . . . in boves irrepsit, Padova 1712; Opera omnia medica et physiologica, Ginevra 1716.

In the "De Morbis Artificum" Ramazzini synthesizes his scientific researches on phenomena apparently different one from another.

Just as the "De Sedibus" has entitled G. B. Morgagni to immortality, so has Ramazzini's De Morbis Artificum raised him by unanimous recognition to the altar of glory.

Were the importance of his work known to the workers of the mine, farm, foundry and other industries as well as to the physician and hygienist everywhere a chorus of praise would be raised to the memory of this famous Italian scientist.

He was designated as the "Third Hippocrates," "glory of the Italian thought and an eminent lighthouse." Castiglione calls him "the first and the greatest industrial hygienist of all times." Koelsch and Pagel state: "to Ramazzini is due the immortal merit of having laid the foundation of a new discipline of medicine."

Ramazzini did not limit his studies to some categories of workmen, or to groups of diseased individuals, because assigned to a specific employment. He extended his observations to all workers, to work itself as a cause prejudicial to the health of those so engaged, arriving at the conclusion that there is no employment which by its very conditions might not be a cause of health disturbance, and that to every type of work there are corresponding morphological and pathological alterations or functional disturbances of a strictly professional nature.

The real merit of Ramazzini consisted in his method of research on the morbid causes and in transferring the study of these causes from the individual to the working environment, from the mode of personal life of the sick workman to the materials associated with his employment, to the methods of work, and the positions and the efforts of the body during a working day. He left the home of the sick to visit the industry that employed the patient and, from the knowledge acquired in the workshop, he pointed the means of collective prevention against disease.

Bernardino Ramazzini was truly the founder and precursor of industrial medicine. Such defender of the physical integrity and health of the worker is now exhumed after nearly three centuries of oblivion to witness a new deal for the workman, as exemplified by Workmen's Compensation Laws, increased interest in occupational diseases, and industrial hygiene.

Bibliography:

DE RENZI, storia della medicina in Itali, Napoli, 1948, IV, passim.

F. KOELSCH, B. R., der Vater der Gewerbehygiene, sein Leben und seine Werke, Stoccarda, 1912.

M. DEL GAIZO, Nel quarto cencenario della nascita di A. Vesalio e nel secondo centenario della morte di B. R., in atti della R. Acc., medico-chirurgica di Napoli (tornata del 3 gennaio, 1915).

A MAGGIORA, L'opera igienica di B. R., in Annuario della R. Universita' di Modena, 1922.

L. DEVOTO, B. R. Biografia, Genova, 1923.

P. CAPPARONI, Profili bio-bibliografici di medici e naturalisti celebri italiani, ecc. Roma, 1925 e 1928, 11 p. 73.

A. CASTIGLIONI, B. R., a Padova in Gaz, degli osped. e delle cliniche, 1927 n. 45.

HERMAN GOODMAN, Medical Record, 1937.

A. PERI, Difesa Sociale, 1938.

Industrial Alcoholism

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THE man would be all right if he would let alcohol alone." I have heard this expression in one form or another for the last 40 years. I have learned during some 10 years' work, largely concerned with a tin plant, and later observed in many other plants during industrial surveys, that workers in certain departments were more prone to drink than those in others, and that those departments were where they were exposed to the breathing of dusts, gases, and poisonous fumes, or to high temperatures usually associated with hard labor. I think most of those who have given thought to the matter will agree with me in this observation. We, of course, have the more or less strenuously applied mental worker who drinks more than is good for him, and with the feeling that it in some way bolsters him for work.

In the investigation of brass foundries, lead

works, painting, and certain other employments during the occupational disease survey of Illinois in 1910, I learned it was oftentimes traditional that it was necessary to imbibe alcohol to prevent the effects of poisons and dusts to which the workers were exposed. Apprentices and young men were so instructed by older employees, although now and then an older employee could be found who opposed the tradition and declared it "a mistaken notion."

Chronic alcoholism among workers appeared to be of such importance that when we conducted the survey of occupational diseases in the State of Ohio, 1913-1915, we made the subject one of the items of standard inquiry on the forms used by all investigators. We used the term *industrial stimulantism* for a broad coverage, implying *alcoholism, coffeeism,* or *druggism.* As a matter of fact druggism was scarcely ever found, but the other two were very prevalent and, naturally, alcoholism received most attention. It was found in numerous industries, trades, and callings, and especially in the types of industries and exposures above referred to. A dust or fume which irritates and dries the throat seems to be especially associated with the desire for stimulants. "It cuts the phlegm in your throat," is a common reason.

In the more domestic field, there is also the tendency toward alcoholic drinks on the part of washerwomen, as well as laundresses in commercial establishments, possibly associated with the vapors or odors of strong detergents and with hot, steamy atmosphere often contaminated with gases and vapors from heating appliances in poorly ventilated quarters.

It is too well known for comment that human beings, when they begin to feel "run down," resort to more coffee, more tea, more alcohol, more "coke," or more use of easily accessible drugs. It may also be contended that where such is associated with exposure to the hazards named, there is good reason for a close relation between cause and effect.

The extent of "stimulantism" in industries is problematic, but statistical studies by Frederick Hoffman some 20 years ago led him to the conclusion that, at that time, about 12% of all industrial employees were unduly exposed to dust, gases, and fumes. While much improvement has occurred in recent years in these industrial exposures, it is probable that millions of workers are still so exposed in this country, while an unknown additional number are subjected to undue heat and fatigue.

Even the weather may be drawn into the picture, but fortunately this is not usually a continuous offender.

Too often one hears that a father or son "was driven to drink because of his work." "He quit the day all in, and naturally began to drink." Of course, many are inclined to scout such an explanation and, in very many instances, underlying disease is the cause and in others plain abandon, or failure of proper mental attitude. But far too often investigation has shown there is something to the contention.

Many have noted, as has the writer, the tendency of workers at the end of the day or shift to strike out at once for the bar or drinking parlor (in old days, the "saloon") on the way home, thus substituting a quickly assimilable food in the form of alcohol when in reality they were hungry and should have eaten a square meal instead. Then, too often, the habit has grown until desire for real food has been submerged and so we have the chronic alcoholic who for many years gets along more or less indifferently as respects his health as long as he works hard, and particularly at manual labor. If he works hard at sedentary work he fattens up and, in the end, contributes to the high rates of morbidity and mortality due to chronic degenerative diseases.

FOR many workers the advice of Haggard and Greenburg[1] is, in our opinion, the real solution; especially his recommendation that fatigue can be prevented and efficiency maintained and increased by more frequent eating while at work. Thus he recommends recesses for lunch periods for many industries. Such, no doubt, would greatly decrease *industrial alcoholism,* if we accept the entity of the term. The more logical thing to do would be to remove the hazards in industry which could possibly contribute to such a condition.

It is interesting to note that in recent years proof has been rapidly accumulating that alcohol does not cause cirrhosis of the liver or Bright's disease or arteriosclerosis, or any other demonstrable tissue damage than its irritating effect on the stomach and intestines, and that its effects are sensorial and symptomatic rather than definitely pathological. Even the attending multiple neuritis and delirium tremens are explained as due to avitamintosis and the lack of other basic food essentials—a pellagra-like condition.[2] But whatever its effects all will grant there is no place in industry for alcoholism.

There are a number of reasons for *stimulantism* in industry. It is not only promoted by subjection to various health hazards and the trade tradition that the use of alcohol is necessary to stimulate and protect against these hazards, but by the absence of good drinking water (easily available), the occasional permitting of drinking of intoxicating liquors while at work, also the all-too-frequent absence of a wholesome welfare attitude, a comprehensive education program and, in general, an industrial efficiency department which visualizes the nature of man as well as the nature of processes.

References:

1. HAGGARD, H. W., and GREENBURG, LEON A.: Textbook, "Diet and Physical Efficiency," Yale University Press, 1935, 180 pages.

2. CECIL, RUSSELL L.: Textbook of Medicine. NEWMAN, HENRY W.: Recent Studies in Alcohol, *Am. J. Med. Sc.,* December, 1936, pp. 868-875. EMERSON, HAVEN: Textbook, "Alcohol and Man," 1932. EVANS, NEWTON E., and GRAY, P. A.: Laennec's Cirrhosis, *J.A.M.A.,* April 9, 1938, pp. 1159-1161. BOLES, RUSSELL S., and CLARK, JEFFERSON H.: The Role of Alcohol in Cirrhosis of the Liver, *J.A.M.A.,* October, 10, 1936, pp. 1200-1203.

Streamlining Medical and Surgical Service

—President's Address: Forty-Ninth Annual Congress of Railway Surgeons, Chicago, September 19-21, 1938—

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IT IS not my intention to recite the medical and surgical accomplishments in the recent past, nor discuss any purely technical subject. We would pay homage and record our respect for those of our members who have labored long and well, enduring the heat and burden of the day, in fostering, developing and producing this Association with its present attainments and prestige, freely admitting it is still in its infancy. We honor them for that which has grown from the seed of their planting. This organization has far-reaching potentialities.

Will you consider with me, for a short while, "Streamlining Medical and Surgical Service" in our great transportation industry, a caption which seems to be appropriate at this time.

The Zephyr, Century and Broadway Limited trains set the pace and establish a record for the new urge now dominant in transportation units. The analogous spirit may well permeate and arouse a similar urge in other coordinated fields of endeavor.

The practice of medicine and surgery has changed and progressed markedly, and there has developed or arisen a new specialist, the result of an obvious need, the Industrial Physician and Surgeon. The triad must be respected in all of our consideration—the industry, the worker and the Doctor. Whether we devote full time or part time, the same basic approach to our problem is necessary; there is no naive aspect to our problem.

Our interest is primarily in human factors and how important it is to assure, from a pre-employment standpoint, that a person is physically and mentally qualified for the position he seeks. We cannot minimize this responsibility when we face the problem of human safety and the individual's right to gain a livelihood. Happily, proper placement often comes to our rescue to assist in solving this problem.

The need is further emphasized to the industrialist by the ever increasing compensation laws, which are intended to protect the employee, and rightly so, in principle. However, industry must not be charged with pre-existing handicaps which cause the individual to be a hazard and a potential liability. The most progressive movement in this particular respect is to refer persons who do not measure up to standard to channels through which remedial measures may be applied to assist him in qualifying and assuming his rightful place in our social structure. I am sure you will agree that this is "Streamlining."

Improvement in service next commands our attention, such as lessened friction, improved bearings—giving comfort, and finally a satisfied clientele. The need of our great fellowship and organization is perhaps expressed best in the exchange of views, the discussion of new methods and the promulgating of a human technique.

Hygiene and other preventive measures in industry are coming to the front on account of the ever increasing introduction in industry of more efficient methods embracing mechanical, chemical and electrical advances. Human toll must not be exacted to establish safety measures.

How evident it becomes that the trained industrial physician is to be a greater factor in all industrial programs, from the human, economic and efficiency aspects.

The safety engineer has accomplished much in protective devices. The chemical and electrical engineers are doing valiant service. We, as human engineers, must evaluate environmental influences and the earliest evidence of disease. We have as agents in this work the most recent laboratory methods, which should always be resorted to in our professional activities. The management is concerned in major problems and it becomes our serious responsibility to formulate a program of procedure in all things that have to do with the health of the employee.

The most recent advance for comfort is air conditioning. Here again, the physician has a task thrust upon him, and, while studies have been made, we are far from a satisfactory solution as to the influence of such artificial atmospheric media on humans. Observation, experience and research will assist in solving this somewhat vexing problem.

Dust diseases are coming to our attention in recent months on account of the emphasis on industrial health. This is such an involved subject that it demands careful research to evaluate properly such alleged conditions, and it is destined to be of no small annoyance to us in the days ahead.

We are advancing in the diagnosing and enforced treatment of blood diseases. There is no yardstick to measure the accomplishments in this field of endeavor.

The heart demands careful consideration, as do the kidneys and central nervous system.

We are in a measure responsible for those who come within our purview and perhaps *particeps criminis* in cases in which we might have assisted in avoiding trouble if we had used ordinary care. Perhaps many present have not given this serious thought, thinking that in the stitching of a wound or otherwise dressing injuries, your responsibility ends. Such I assure you is not the case.

Many are the changes and advances in the field of surgery, and the importance of keeping abreast of the times is evident. Injuries to the head, back and abdomen, including fractures, call for the best

our profession has to give. We cannot go into details, but suffice it to say proper care early will lessen the need of reconstructive surgery later.

STREAMLINING calls for the shortest period of disablement, the best restoration of function and the returning of the individual to economic security. I am not particularly interested in getting the man back to work to avoid reporting case to I.C.C. when such efforts involve a hazard. Perhaps the I.C.C. will some day make reportable injuries according to mutilation rather than lost time, thus removing possible collusion.

The streamlined train transports the passenger quickly, safely and comfortably, and the destination is reached in a happy mood. Can we do less? We have found there is a human technique in the handling of our accident and alleged accident cases which brings large financial results to our railroads, not only in the increase of business, but also through the claim department, making settlements more reasonable and quickly adjusted.

There is frequently encountered the Doctor who, in litigation or forced settlements, will make statements which are completely reversed after settlement. May the time come when such deliberate acts will be properly handled by his fellow colleagues. I am glad to say, in my opinion, this is not a growing but rather a diminishing abuse of professional privilege.

At the end of the first run of the new Broadway Limited, on which I rode, it was gratifying and pleasant to hear the passengers remark on what a pleasant trip, how smooth and fast we ran; with all the luxuriousness one could wish.

It is equally pleasant to hear the employee say: "Doctor, I want to thank you for your thoughtfulness and kindness at all times, and especially during the treatment of my recent accident. I am now 100% and it is all due to your splendid and wise care."

Is that not developing a streamlined service with a satisfied clientele?

IN CONCLUSION, we have made advances in the activities of our Association as follows:

1. The development of individual initiative and its encouragement.

2. The promulgating of professional industrial accomplishments for the good of all.

3. The normal relation of Doctor and patient including a human technique.

4. Proper respect for all interested—employer, employee and public. Such a relation, if continued, will go forward in leaps and bounds and will not be self consuming.

There were three wise men crossing the desert and they came to a spot where there were a group of bones. And there they paused. One wise man said: "In our country we have the power to say the word and the bones will be restored to their normal relation." The others told him: "Say the word." He did so, and the skeleton appeared. The second wise man said: "In our country we can say the word that will endow the skeleton with flesh, and arteries, and skin." They told him: "Say the word." He did, and it was so. The third wise man said: "In our country we can say the word and life will be fully restored." The others urged him to say the word. He said it, and life appeared in its fullest form. Then, behold! The lion ate all three men.

May we not unwisely contribute to produce a state of affairs that will doom us.

Individual initiative must not be curtailed, fitness and ability should be the only guides in selecting a medical personnel, and a continuum of this great fellowship and of our associational activities will preclude the possibility of an octopus arising in our midst to consume us.

Mental and Emotional Instability

—A Problem of Late Midlife—

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MY GOOD friend and one-time preceptor in medicine, Dr. Sterling B. Taylor of the New York Central System, to whom I am indebted for the honor of appearing on this program, first suggested the subject of this discussion to me.*

To some mental instability may seem to be a subject of minor concern to a Congress of Railway Surgeons whose routine duty requires them to be familiar with the latest advances in allergy, arthritis and cardiovascular disease but who must be prepared on a moments' notice to take charge of an amputation, a broken back or a ruptured appendix.

Whether he realizes it or not, no surgeon charged with determining the physical fitness of the men who man the great railway systems of the country would hold his position long if he did not take into consideration the mental and emotional element in his examination.

The marvellous advances in the comfort, speed and safety of train service have not lessened the importances of the human equation. In spite of ever increasing automatic safety devices, the man who controls the throttle must have a good eye, a ready response, a clear head when he thunders over a grade crossing at 70 or more miles an hour. He must be a better man, mentally and physically, than his father or grandfather who made the same run a generation or two ago.

The mental fitness of a man is dependent upon many factors. The fundamental ones such as heredity, constitutional make-up, general health, etc., have all come under consideration in railway service long before he reaches midlife. We are here concerned with the variable factor, mental and emotional instability, which represents an inefficient response on the part of an individual to his experiences, what he feels, and sees and thinks, all

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of which combine to make up our emotional state.

To illustrate, no engineer can be at his best on the run when he has just left his only child's bedside after a mastoid operation. No fireman can give his undivided attention to his duties with papers in his pocket notifying him that his wife is suing him for divorce. No conductor can fail to reflect some of the irritability he feels to passengers if he is losing sleep waiting on a sick one at home. Nor are the effects of an evening of dissipation removed by a few hours of sleep. All of these causes and many more operate daily to lessen the efficiency and lower the safety factor in the remarkable organizations we serve as doctors.

But we are interested in some of the more tangible causes of emotional instability in the age group between 50 and 65. There are a number of pathologic conditions deserving of emphasis for we must recognize that any physical disease can produce some of the symptoms which include easy mental and physical fatigability, increased irritability, poor emotional control, difficulty in thinking and often impaired memory. It may require a careful investigation to determine whether it is arteriosclerosis or worry over impotency, a brain tumor or anticipation of being placed on the pension that provokes the faulty cerebration. I will refer to a few organic causes first.

CASE 1. A veteran conductor eligible for retirement in five more months. He had been in failing health for years. His son-in-law brought him to my office with the complaint that he couldn't sleep, was nervous, irritable and forgetful. He thought a nerve tonic might help him last out these last few months. Recently he had been having some peculiar spells, his complaint was of dizziness but he rubbed his right arm a lot. His speech was thick for several hours and he had difficulty in finding the right word. His blood pressure had been high for years but the last few times the doctor said it was normal.

It is not necessary to wait until paralysis occurs to make a diagnosis of arteriosclerosis. Nor do all cerebral hemorrhages and thromboses produce a paralysis, either motor or sensory. Too often the acute indigestion which is accompanied by dizziness, numbness, weakness and other vague symptoms represents a cerebrovascular accident and while definite localizing signs may not occur there may be sufficient scarring to produce chronic symptoms. A restricted blood supply due to changes in or around the arteries of the brain inevitably limits the amount of available oxygen which is vital to the functioning of the cortical cells. Disturbed function is reflected in the headaches, dizziness, irritability, forgetfulness and anxiety, to mention only a few of the common symptoms.

Arteriosclerosis is peculiarly a problem of railway employees. The demands of their work are such as to provide stress and strain on the cardiovascular system and the more susceptible body types are the ones best fitted to meet the demands from a physical standpoint. Again, in most vocations as a man approaches 60 he can begin to take it easy, younger men are expected to do the hardest jobs. In train service at this age a man is apt to be on his fastest run, at his maximum earning capacity, and even the prospects of a pension are not pleasing under these circumstances.

CASE 2. A clerk in a freight office, just past 50, came to me privately because he wasn't able to remember "like he used to." He had been bothered with some dizziness and a few headaches, and his friends remarked that his speech wasn't as plain as it ought to be. He was sure it wasn't his false teeth. He wanted a complete check-up.

The possibility of an early arteriosclerosis at once suggested itself. However, the blood pressure, and the retinal and peripheral vessels were all apparently normal. The added description of what appeared to be several brief convulsive attacks renewed the thought of cerebral arteriosclerosis. Arteriosclerosis should always be considered along with brain tumor when a convulsive seizure occurs for the first time after 40.

The neurological findings afforded a clue. When the man stood with his feet together and his eyes closed he was quite unsteady. His knee jerks were diminished. There had been some difficulty in starting his water. The pupils were not only irregular but reacted poorly to the light of a strong flashlight held off to one side to eliminate accommodation to distance. He found it very hard to repeat Methodist Episcopal three times. The spinal fluid confirmed the clinical diagnosis of neurosyphilis with beginning signs of brain and cord involvement. He had been treated for syphilis during his service in the A.E.F.

CASE 3. Some time ago two brothers, both enginemen, brought another brother, only 47 to me because of alcoholism. For many years he had been a mechanic, dependable, well-liked and able to get along with everyone in the roundhouse. Several years before he had struck his head while inspecting a boiler but had paid little attention to a minor injury. Soon afterward, it was claimed, he began to show a change of attitude, became irritable, unreasonable, emotionally unstable. He began to drink and his wife divorced him. He continued at his job although his foreman recognized that he was not himself. He finally laid him off, advising him to see a doctor although it was believed among his friends that his domestic troubles had driven him to drink.

The alcoholism was an obvious cause and he readily admitted he had been drinking steadily for over a year. Chronic alcoholism can produce a clinical picture known as pseudoparesis and clinically indistinguishable from paresis. The history of the injury suggested the possibility of a chronic subdural hematoma, a walled off collection of fluid of high protein content which could act as an expanding tumor in the brain cavity and produce a gradual change in his personality. Again the telltale pupillary responses of Argyll Robertson, the inability to say Methodist Episcopal, the grandiose ideas even when he was sober pointed to the need a confirmatory lumbar puncture and fever therapy followed by tryparsamide and bismuth.

Less than 40 years ago at least one professor of psychiatry in this country attributed paresis to the

vibrations of train travel. He had observed it so frequently in engineers and firemen, not to mention other men in train service and *"post hoc propter hoc."* Paresis is still to be thought of when a man of good record begins to show character changes, when former ideals and standards are replaced by carelessness, when recklessness, forgetfulness, and personal indulgence appear without the individual showing much appreciation of the changes which are obvious to relatives and friends.

CASE 4. The next patient presents a study in mental instability. A man of 55 years, a trainmaster of the best standing, his wife consulted me in regard to having him declared insane. From her attitude I suspected the existence of "another woman" in the case but not the kind she described to me. On an auto trip he had picked up a hitchhiker 19 years old, unattractive and common in every sense of the word. Within a few weeks he had announced to his wife, his son and daughter, both students in the University, that he proposed to live with his new found friend and suggested a nice quiet divorce.

At my suggestion the wife inquired of his superiors and found that his work was quite satisfactory. However, her refusal to divorce him soon led to an ultimatum—"give me a divorce or I'll resign my job." He later told me that he intended to anyhow—to move out on a small patch of ground, in an old, run-down shack to raise sweet potatoes, peanuts and hogs, though he had never lived in the country in his life.

Briefly, the following happened. To my surprise the man willingly consented to a complete examination. Instead of the worn out, deteriorating man I expected to see he came well dressed and apparently in the prime of life. He admitted the circumstances were strange, and that it must look crazy to others, volunteered that he was living without the benefit of clergy, said he might be arrested on a delinquency charge which his wife threatened to bring but insisted that he was doing exactly what he wanted and intended to do no matter what happened.

He insisted that there was nothing wrong with his mind, that he had always wanted to live the simple life, that his wife had driven him on and on from one promotion to another, to higher pay, more responsibility—all to enable her to entertain her clubs, dress better, put the children through college, live in a better neighborhood than they could afford. Now he had rebelled. His children were ready to graduate, the home was paid for and in his wife's name. She could have their savings but he wanted his freedom.

The physical and neurological findings were entirely negative. The blood and spinal fluid were negative. The two physicians appointed to examine him by the court found him sane. An apparently normal man, still in the prime of life had turned his back on his family and home, his job and friends, all that makes for material success to live with one who looked up to him instead of correcting his English and criticizing his dress and manner, who made him feel necessary to her happiness in life, and who revived his waning sexual powers, who lowered the demands of his environment to levels he could maintain without effort and in relative comfort. Several years later he is still content according to my information.

Some will doubtless contend that the patient just described must have cellular or organic changes in the brain whether they can be demonstrated or not. Others will prefer to group him in the functional disorders — personality changes where the underlying pathology may be physiological and reversible rather than structural and therefore more or less final. With this in mind let us consider the following.

CASE 5. A research engineer in the automotive industry rather than the railroads, famous for inventing and perfecting a major advance, yet he described himself as a complete failure. He complained of insomnia, of crying or weeping at the most inopportune time, of indecision in the most trivial matters such as the choice of his tie for the day. He confided to his wife that he had committed the unpardonable sin, that suicide was the only way out.

He was depressed and looked it. He was slow in his responses, both physical and mental. He was evidently as ineffectual as he felt. Yet the physical findings were essentially negative. The tongue was coated, the breath bad, his bowels sluggish, he had lost some weight. His metabolism was within normal limits although he had stopped taking the thyroid prescribed at the onset of the illness several months before. Other laboratory tests were not remarkable.

The past history, taken chronologically, told the story. As a child there had been a certain moodiness. While still in college he had lost appetite and weight, had been sent to the country for a rest, and lung trouble had been mentioned. He came back stronger than ever, in fact he always did after his illnesses. He did exceptionally well for several years until he began to be heart conscious, lost interest in things about him, began to worry about himself and finally had his tonsils removed. He recovered after a number of months. Following his marriage he succeeded remarkably well—perfected several inventions and again his health failed. This time the thyroid was suspected. He was ill for a number of months after the operation. He recovered completely and soon had found a new interest in his work which carried him at top speed and high tension until about six months before I saw him when he began to slow down and complain.

The ups and downs of the cyclothymic individual whose biological rhythms are too marked to take in one's stride produce a specific kind of mental and emotional instability which may not disable in the milder forms but which may produce one of the most characteristic of the psychoses, the manic-depressive, with its alternations between over and underactivity, between too good and too bad feeling, between flight of ideas and mental retardation. Sometimes only one of the phases appears and not infrequently only one attack of importance occurs during a life time. In its milder forms it produces an emotional instability which

is hard to recognize without the background of the past history. These periodic depressions must always be considered when confronted with an individual who swings from one extreme of affect to the other, full of energy and ideas or more or less dormant, or expending a minimum of psychic and physical effort.

In this connection we should mention involutional melancholia which occurs in men between 50 and 65 years of age, and corresponds with the menopausal disturbances in women at a somewhat earlier age. It represents the mental and emotional reaction of a certain type of personality to the chemical and glandular changes of the period. The susceptible person has usually been of a somewhat pessimistic nature, over-conscientious and adhering outwardly to a rigid standard of conduct although more often than not secretly wishing to ignore them, an individual whose interests were narrow, his friends few, inclined to be saving and close. The individual develops symptoms of emotional instability with restlessness, poor appetite, loss of weight and sleep, becomes tense and anxious, makes unreasonable physical complaints in explanation of his illness, develops fixed ideas of an hypochondriacal nature and yet continues quite capable of an occasional smile, may even go on working at some routine job although fully convinced that his bowels are stopped up and "it is only a matter of time." Involutional melancholia differs from the manic depressive disorder in the absence of previous attacks, in the characteristic tension and anxiety which are present and the personality pattern on which it develops.

With one more group illustrated by another case I will end my clinic. The patient, a claims adjuster, came to the office at the suggestion of a mutual friend, a doctor. His wife was, at the time, a patient in a hospital and was about to have her second breast removed. He had been told it was malignant, that the chances were not good. Overnight he became acutely ill, developed an acute anxiety state with palpitation, nausea, vomiting, diarrhea and fear that he would commit suicide.

Patients in acute anxiety are often safer in a hospital, particularly if the doctor intends to do any probing of the unconscious for possible psychic irritants to explain the illness. In the case in question the following facts soon came to light. He married early and the responsibilities for the support of his wife and the two little girls who arrived in the next few years weighed heavily upon him. His feeling of inadequacy he concealed by assuming a certain gruffness which his friends referred to as "all business." His family thought it meant he wished to be left out of their trivialities, with resulting disappointment, later resentment, on his part. While they lived as man and wife for 25 years the mutual understanding was far from satisfactory—he unconsciously hated his wife for the wall that had come between them—for the sexual isolation he had actually assumed of his own accord.

The operations and the doctor's prediction of only a few months to live on the part of his wife aroused the repressed feelings of resentment and unconscious hate and brought them too near the surface of consciousness for comfort. Under the circumstances these aroused secondary feelings of terrible guilt and the fear that he would punish himself. It was an intrapsychic conflict, a war within himself. Many patient and sympathetic interviews followed before he could discover for himself the obvious and face the facts.

It is certainly in order to point out that here as in all the neurotic mechanisms, whether neurasthenic, hysterical, anxiety or psychasthenic there are certain compensations in the symptoms for the sick one. By taking flight into illness, the unstable or neurotic personality temporarily and sometimes permanently finds refuge from the problems of life. Certain other individuals, faced with the same situation resort to alcohol and drugs. We all find briefer and more acceptible escape when our situation approaches the intolerable by resorting to an evening out, a game of golf, a vacation trip, yes, even a convention.

In approaching the subject of mental and emotional instability in such broad and general terms I realize that I have not contributed to your knowledge any new facts nor deeper understanding of the subject. It has been my purpose to review some of the more common causes of mood disturbances by presenting a few clinical cases. Reversing our previous order we can recognize three main groups of depressions:

(1) The neurotic in which I would include the transient or reactive depressions — the anxiety states—the neurasthenic and psychasthenic states as well as the hysterias; (2) the periodical depressions which seem to have biological or constitutional basis; (3) the involutional melancholia which occurs quite often in its milder forms in individuals of a susceptible personality makeup. In the organic group while brain tumor, encephalitis, the traumatic constitution and other causes are to be considered, early senility, arteriosclerosis and syphilis provide the majority of the real problems in my experience and certainly are to be considered in every differential study.

To these problems of mental medicine, formerly outside the province of the railway surgeon, the newer problems of preventive medicine and improved safety measures for men in railway service compel your interest and study.

Milwaukee Association of Industrial Physicians and Traumatic Surgeons

THE industrial men in the Milwaukee area recently organized the Milwaukee Association of Industrial Physicians and Traumatic Surgeons. The new Association is a component of the Central States Society of Industrial Medicine and Surgery, and will meet with that Society at the latter's Annual Mid-Winter Clinic on December 9, 1938, at Milwaukee.

Light, Vision, and Seeing

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General Electric Company, Nela Park, Cleveland

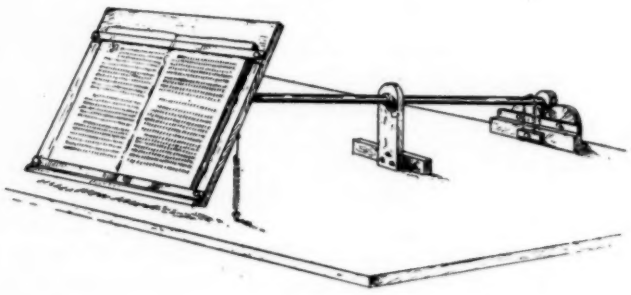
SEEING is more than visual function.* It is a universal activity of human beings and is intimately responsible for their efficiency, comfort, safety and welfare. As such, we regard it as a task requiring the expenditure of effort and energy in its performance. In this respect, it may be compared to other human tasks which utilize human resources and which may be performed with efficiency and ease or with uncertainty and difficulty depending upon the conditions under which the work is done. The present discussion is concerned with effectiveness of various controllable aids to seeing and with the research techniques which have been applied for the appraisal of the effectiveness of such aids to seeing. Since these phases of the science of seeing[1] are both extensive and complex, this discussion is restricted by space limitations chiefly to a consideration of light and lighting which are universally controllable factors.

However, the influence of other factors upon visual efficiency may be inferred from these discussions.

Obviously, the visibility of objects to be seen and the certainty, speed and ease with which they are seen is enhanced as the illumination and the lighting are improved. It also may be assumed that the extent to which light is utilized for these purposes will depend largely upon the significance of the visual benefits obtained from an improvement in lighting which is economically possible and technically practicable. These facts are generally ascertainable only from carefully controlled laboratory researches. It is not to be expected that they can be demonstrated in individual situations any more than it could be expected that the physician could quickly demonstrate the effectiveness of a given vitamin. However, the research basis for lighting recommendations is a matter of utmost importance to those who eventually may appraise or prescribe the visual conditions under which human eyes must function.

In the performance of visual work it is usually assumed that speed and precision are appropriate criteria for appraising the benefits obtainable from any aid to seeing. In fact, these criteria have been extensively employed in numerous so-called "practical" investigations of the influence of light and lighting in industrial situations. However, it is now known that these criteria are adequate only in those cases in which the visual demands of the task are the limiting or governing factors. For example, the speed or precision of performing many manual tasks which are guided by vision is often limited more by mechanical than visual factors. In other situations, such as reading, the speed of reading may be limited by the mental processes and habits rather than by the functioning of the ocular mechanism or visual sense. An experimental basis for this conclusion is cited.

In Fig. 1 is illustrated an arrangement for determining the speed of reading under two conditions: (1) with the printed matter held in a fixed position; and (2) with the reading matter rapidly vibrated in a non-uniform manner. Under the latter condition the task of reading was so difficult that the eyes and subjects were severely fatigued by the reading of two pages of well-printed type. Notwithstanding this fact, the



READING MATTER	READING TIME	PROBABLE ERROR
STATIONARY	1.51 MINUTES	0.04 MINUTE
VIBRATING	1.57 "	0.05 "

Fig. 1.
Illustrating an experiment which showed the ability of the human seeing-machine to compensate for poor visual conditions. The data indicate that there was little difference in the speed of reading with the printed matter vibrating or stationary, even though the visual difficulty was enormously greater in the former case

average time required to read the two pages was substantially the same under the stationary and vibrating conditions. These data clearly reveal the inadequacy of the criterion of speed of reading as a means for appraising ease of seeing. Thus it is not reasonable to assume that this experimental criterion would invariably indicate the advantages of better lighting. However, it follows from theoretical considerations as well as from direct experiment, that significant differences in speed of reading may be obtained when the reading is continued almost to the limit of endurance of the subject. Obviously, the latter technique is neither desirable from a research viewpoint nor are the results obtained under such abnormally severe conditions particularly significant in everyday practice.

The performance of industrial tasks under normal or usual conditions for extended periods often reveals the advantages obtained from improved visual conditions.[2] For example, the data of Fig.

* Presented at the Twenty-Third Annual Meeting of the American Association of Industrial Physicians and Surgeons, and Midwest Conference on Occupational Diseases, Chicago, June 6-9, 1938.

2 show the increase in production obtained in a statistical operation known as key-punching due to (1) an increase in the level of illumination upon the work from 8 to 60 footcandles, and (2) to a change in the position of the copy with respect to the machine so that it could be read with the eyes in their normal or primary position. In general, it will be noted from Fig. 2 that production began to increase and errors diminish almost immediately after the installation of the new equipment. However, it is of interest to note that these changes did not reach their optimum values until about 18 months later.

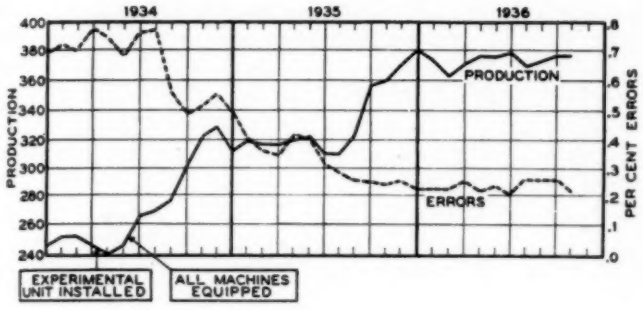


Fig. 2.
Showing the increase in production and the decrease in errors, respectively, which followed the installation of supplementary lighting units upon business machines

These experimental results emphasize the complex nature of human behavior with respect to environmental changes. Thus it seems rather naive to assume that mechanical and visual habits acquired from years of training will necessarily be greatly or at least completely altered immediately upon changing the visual conditions under which the work is done. For similar reasons, it is often necessary for a patient to wear properly prescribed ophthalmic corrections for some time before he acquires the maximal benefit from them. The fact that the improvement in the performance of the key-punching task was almost negligible even after a month of work under the new conditions may be considered as evidence of the ambiguity of so-called "production tests" of short duration.

Since it is the consensus that eyestrain is largely of a muscular character, it is possible that the influence of illumination on ease of seeing might be indicated by measurements of convergence reserve taken before and after a period of visual work under different levels of illumination. For this purpose, the authors designed the convergence ergograph illustrated in Fig. 3. In general, the results of an extensive investigation involving the criterion of extrinsic muscle fatigue indicated decrements in the amplitude of convergence reserve of 3.0 and 1.0 diopters after reading for one hour under one and 100 footcandles, respectively.[3] Therefore, the assumption is made that muscular fatigue produced by reading, as indicated by changes in the tonicity of the internal recti muscles, is approximately three times as severe after reading for one hour under one footcandle as it is after an hour's reading under 100 footcandles. Somewhat similar results were obtained by measuring the amplitude of convergence reserve before and after the day's work in the key-punching operations previously discussed.

It is a matter of every-day experience that nervous tension and fatigue cause a tightening of certain muscles. For example, a pencil may be held more tightly when one is nervous or after the muscles of the hand and arm have become fatigued from a long period of writing. Hence it was conceivable that appropriate measurements of nervous muscular tension[4] would indicate certain differentials in the subtle psycho-physiological reactions to light, lighting and other factors which influence the performance of visual tasks. The technique which we evolved to obtain an objective indication of tenseness consisted in measuring the changes in muscular tension as registered by fingers of the left hand which rested naturally and comfortably upon a large flat knob which was mechanically attached to apparatus suitable for recording the pressure exerted upon the knob. This mechanism was concealed from the subject, and during a period of about a year in which the investigation was in progress, the subjects were unaware of the fact that the pressure they unknowingly exerted upon this knob was being recorded.

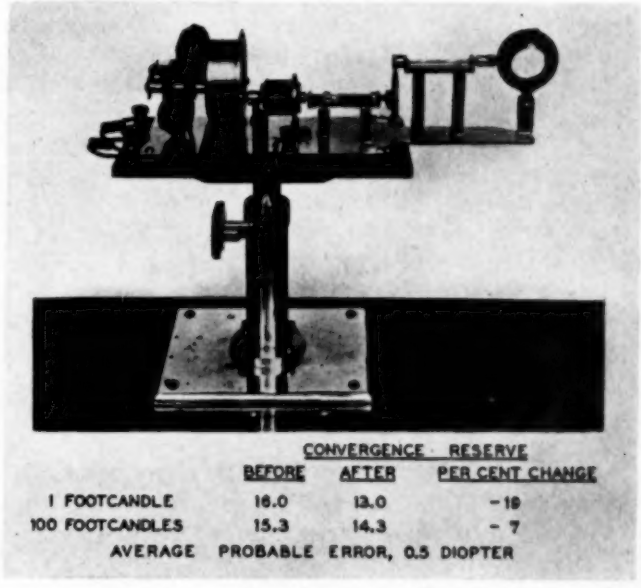

CONVERGENCE RESERVE
BEFORE AFTER PER CENT CHANGE
1 FOOTCANDLE 16.0 13.0 -18
100 FOOTCANDLES 15.3 14.3 - 7
AVERAGE PROBABLE ERROR, 0.5 DIOPTER

Fig. 3.
The convergence ergograph designed by the authors

The subjects were instructed to read at their natural rates during the 30-minute test-periods conducted under each of three levels of illumination—1, 10 and 100 footcandles, respectively. The experimental results are summarized in Fig. 4 for each of the 14 subjects and for the entire group. It is apparent from these data that the differences in the physiological reactions resulting from reading under various levels of illumination are of such definiteness and magnitude that they are capable of creating muscular strains which are measurable by the method described. Hence

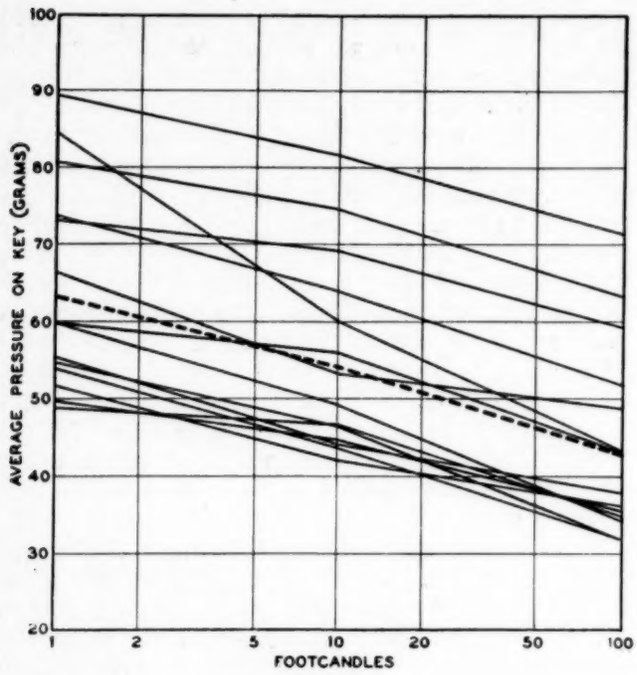


Fig. 4.
The relationships between indicated nervous muscular tension and level of illumination for each of the 14 subjects. The broken-line represents the geometrical mean of the results of the 14 subjects. The probable errors of the average values of nervous muscular tension are less than 1%

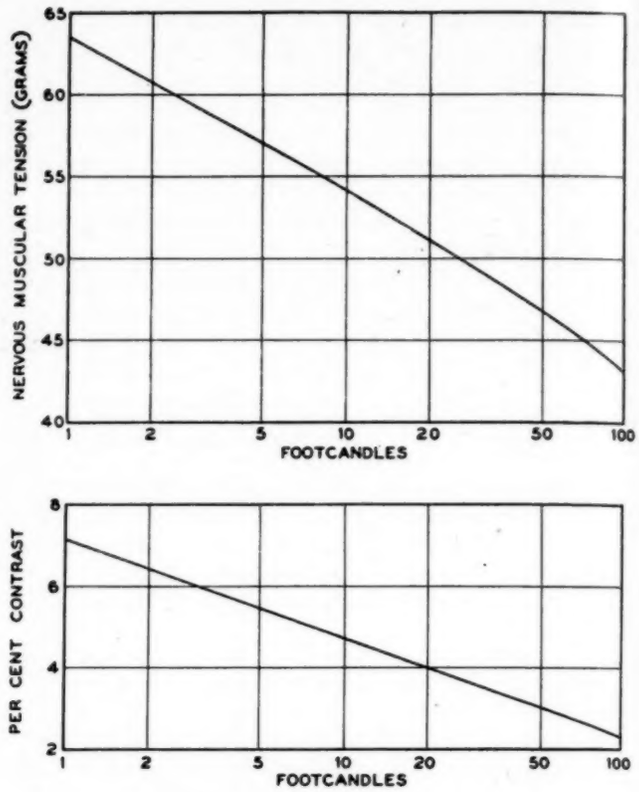


Fig. 5.
The upper plot shows the relationship between level of illumination and indicated nervous muscular tension induced by reading. The lower plot shows the relationship between level of illumination and the minimum contrast required for threshold visibility of an object equivalent in size to the book-type used in the tenseness investigations. Both relationships are generally logarithmic in character

it seems reasonable to assume from these results that the decreased tenseness concomitant with reading under the higher levels of illumination is an indication of favorable visual conditions from the viewpoint of ease of seeing. Thus experimental evidence is now available which indicates that not only is visual efficiency increased through the use of higher levels of illumination but also that this benefit is obtained with a decreased expenditure of human energy. The correlation between these relationships is shown in Fig. 5, the upper curve of which shows the average decrease in nervous muscular tension while reading under illuminations from one to 100 footcandles. The lower curve shows the reduction in contrast required for threshold visibility of objects equivalent in size to the type read in the tenseness experiment. The same range of illumination is involved in both cases. It will be noted that both relationships may be represented by approximately straight lines when the footcandle values are plotted logarithmically. Thus it is evident that the correlation between the psycho-physiological *effects* of seeing and *visual function* is an extremely close one. Therefore, ease of seeing may be appraised, at least relatively and approximately, by measurements of visual function or visibility.

In view of the significance which has been attached to the relationship between illumination and tenseness induced by the performance of a critical visual task, it seemed to be advisable to apply other criteria as possible checks upon the foregoing conclusion. Since the heart-rate is influenced by numerous physiological and psychological stimuli, it seemed possible that the expenditure of human energy in critical seeing might also be revealed or indicated by changes in the heart-rate.[5] Thus the initial purpose of our researches upon the heart-rate was to seek additional experimental evidence of reflex effects inaugurated by critical seeing. The influence of visual effort upon the heart-rate is clearly indicated by the relationships presented in Fig. 6. The important facts revealed by these data are (1) that the decrement in the heart-rate was much greater under one footcandle than it was under 100 footcandles and (2) that the decrement in the heart-rate appears to be due to the effort of reading rather than to mere photopic stimulation. It is quite possible that the gradual decrease in the heart-rate is due, in part, to the fact that the subject remained seated for a rather extended period. However, this factor may be assumed to be constant under both levels of illumination. It will be noted from Fig. 6 that the heart-rates at the beginning of the first reading-period, which immediately followed the first rest-period, are essentially equal, and that the rates changed rather abruptly when the subjects began reading. This result is obvious in both the first and second periods of reading. Thus the definitely lower heart-rate under the lower level of illumination represents additional evidence of reflex effects from reading under presumably unfavorable lighting conditions. The sequence of the two levels

of illumination was reversed in half the total test-periods for each subject. It is obvious that the same results were obtained.

From our viewpoint, a depression of the "normal" heart-rate due to critical near-vision for prolonged periods under conditions unfavorable for seeing, may produce significantly deleterious effects—not indeed upon the heart itself, but upon other bodily functions. For example, such reflex effects might inhibit the processes of digestion and defense to some extent; and the integrated effects of such inhibitions over a period of years might be a factor of no little importance. It does not seem unreasonable to philosophize that the visual and neurological mechanisms in man have been developed through evolutionary periods for other purposes than prolonged concentration upon highly critical and exacting visual tasks. These are artificial tasks imposed by modern civilization without regard to their eventual physiological consequences. Furthermore, we are inclined to consider other sensory stimuli as possible sources of such unfavorable reflex effects. Much has been written, for example, regarding the effects of noise upon human welfare. Statistics on human morbidity and mortality certainly indicate that penalties are being paid, even though the causes may not be obvious or established.

The basic conclusions relating illumination and ease of seeing which were derived from the criterion of nervous muscular tension are also supported by the results of researches involving the frequency of blinking as a criterion of ease of seeing. In general, a consideration of the possible physiological causes of the normal movements of blinking leads rather directly to the viewpoint that these movements are related to the degree of tension or state of fatigue of the subject. As stated by Ponder and Kennedy[6]—"the rate of blinking is closely related to the mental tension of the subject at the time and that the movements constitute a kind of relief mechanism whereby nervous energy, otherwise unutilized, passes into a highly facilitated path." This theory is given support by the results of our investigations in which the frequency of blinking was determined during the performance of critical visual tasks under favorable and unfavorable conditions for seeing. In fact, the results obtained by us in using this criterion indicate that it is one of unusually high sensitivity to physical changes in the stimulus.[7]

We have shown that the following changes in the visual situation increase the rate of blinking:

1. An increase in the duration of the period of reading.
2. A decrease in the size of type read.
3. The use of improper eyeglasses while reading.
4. The addition of a glare-source within the visual field.
5. A decrease in the level of illumination upon the reading matter.

It is obvious that these changes in the visual situation are unfavorable ones from the viewpoints of clear and easy seeing. The actual experimental results obtained in our researches are shown in Fig. 7. It will be noted, for example, that the rate of blinking is much greater when the reading was done under one footcandle than under 100 footcandles. In other words, these data indicate that reading becomes easier as the illumination is increased from one to at least 100 footcandles. The same conclusion also follows from the criteria of visual function, nervous tension, fatigue of convergence, and others. Some of these experimental results are summarized in Fig. 8. We have also applied the criterion of rate of blinking with apparent success in the study of design of type, leading of type,[8] printing on tinted papers,[9] etc. The results correlate nicely with

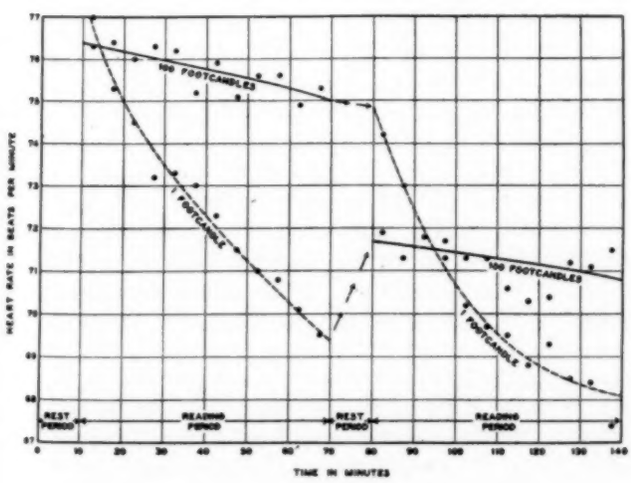


Fig. 6.
The relationship between heart-rate and duration of the reading for each successive five-minute interval of the entire test-period. Each plotted point represents an average of 70 test-periods. The probable errors of these data are generally less than 0.5 of a beat per minute

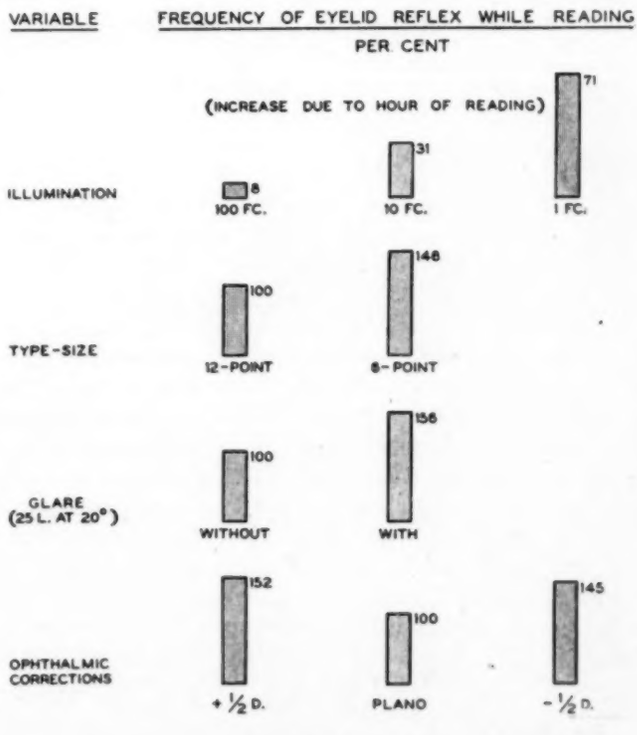


Fig. 7.
Changes or differences in the frequency of blinking while reading under various visual conditions

measurements of visibility in a logical manner.

The data of Fig. 8 may be considered to pertain, in general, to the influence of illumination upon (1) visual efficiency as appraised by momentary measurements of visual function such as visual acuity and contrast sensitivity, and (2) upon ease of seeing as appraised by the integrated physiological effects of strain and fatigue. It follows from the close correlations among these seven

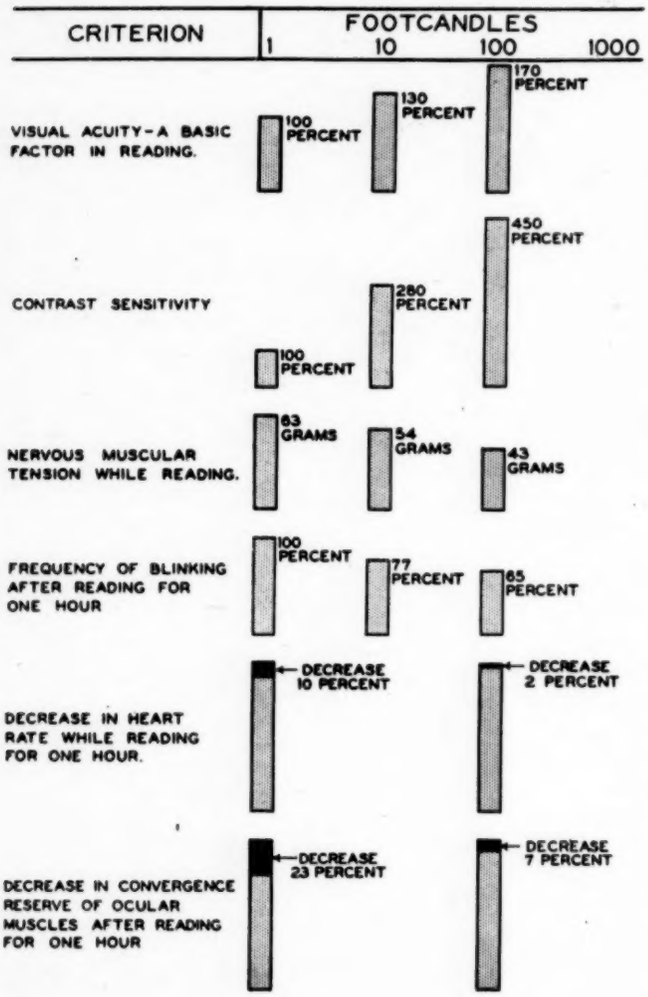


Fig. 8.
A summary of results of extensive researches which reveal the influence of illumination upon important aspects of visual efficiency and upon even more important effects of seeing. Researches dealing with these and other effects are revolutionizing the viewpoint from barely seeing to easy seeing

diverse criteria (and others) that the favorable influence of illumination upon the efficiency of the visual processes and upon ease of seeing extends to levels of illumination at least as high as 100 footcandles. Furthermore, since the backgrounds in all cases were white surfaces, the foregoing conclusion is equivalent to stating that increased beneficial visual effects are obtained for brightness-levels at least as high as 100 footlamberts.*

It will be noted from Fig. 8 that the illumination must be increased in geometric steps to obtain arithmetic increases in the measured visual effect. Similar relationships between stimulus and sensation are encountered throughout the realm of sensory physiology. This is an unassailable fact which did not permeate far into considerations of light and lighting until recently. It is a characteristic of visual response which often leads to naive conclusions. For example, the statement is not infrequently made that significant improvements in seeing are not obtained for levels of illumination higher than some arbitrarily conceived value. In general, the latter conclusion is usually a result of a confused compromise between economic factors and visual benefits. Obviously, the conclusions derived from psycho-physiological researches pertaining to the influence of illumination upon visual efficiency and ease of seeing are in no way related to economic factors. The latter only become involved when recommended values of illumination are specified for various visual tasks.

Fig. 9.
The scale of footcandle effectiveness. Numerous and diverse researches have established the fact that the illumination (footcandles) must be doubled to produce equal and significant improvements in seeing

If the ability and performance of the human seeing-machine were constant, rather small changes in illumination would noticeably affect seeing. However, the effectiveness of the visual processes varies from moment to moment due to involuntary fluctuations of the "focus" of the eyes and of the attention, and to other human vagaries. In general, analysis of the magnitudes of such fluctuations and introspective considerations of visibility and ease of seeing lead to the conclusion that the level of illumination must be approximately doubled to produce an obvious and significant improvement in seeing. This becomes a cardinal principle in consideration of footcandle recommendations and Fig. 9 is a graphic illustration of it. This does not mean that one footcandle added to 10 footcandles, for example, will have no influence on seeing; but rather that this influence will be very small and that it will not be detectable by the unaided human seeing-machine.

Our researches relating illumination and seeing have generally been confined to the range from one to 100 footcandles. This is equivalent to six successive steps on our *footcandle scale of effectiveness* as will be noted from Fig. 9. These may be regarded as successive steps toward ideal lighting from the viewpoint of ease of seeing. If a given visual situation is to be improved through the use of a higher level of illumination, it follows that at least one step is essential; others are desirable but may not be recommended in certain cases, due to factors of cost. In considering the higher levels of illumination it is advisable to give some thought to the system of lighting. For years

*The **footcandle** is a unit of illumination. It is approximately the illumination on a small area of a vertical surface one foot from an ordinary candle.
The **footlambert** is a unit of brightness. It is the brightness of a perfectly white and diffusing surface which is illuminated to 1 footcandle.

we have advocated consideration of general illumination plus supplementary or directed lighting.

The correlation between measurements of visibility and ease of seeing greatly enhances the significance of the former as a criterion for appraising the effectiveness of various controllable aids to seeing. Such measurements are readily obtainable in a wide variety of practical situations through the use of the Luckiesh-Moss Visibility Meter.[10] This instrument, illustrated in Fig. 10, consists essentially of two colorless photographic filters with precise circular gradients in density which may be rotated in front of the eyes while looking at an object or performing a visual task.

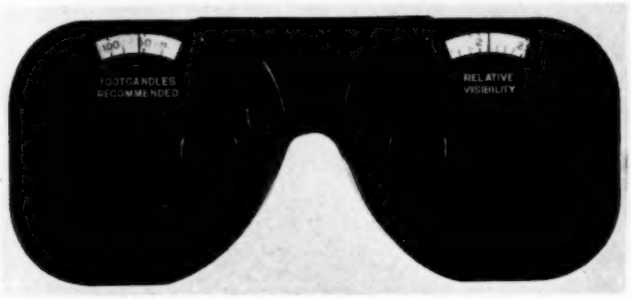


Fig. 10.
The Luckiesh-Moss Visibility Meter. A new instrument for appraising the visibility of various objects and visual tasks and for specifying practical levels of illumination for visual tasks upon a rational basis. The illustration shows the side of the meter towards the eyes

These gradient filters or circular "wedges" not only reduce the apparent brightness of the visual field due to absorption but also lower the contrast between the object of regard and its background due to a scattering of light by the photographic film.

A scale value of "1" for this instrument indicates that a simple "black" object upon a "white" background, whose critical detail subtends a visual angle of one minute, is of threshold visibility when viewed under an illumination of 10 footcandles by a person possessing normal vision, through the corresponding region of the filter. This is the accepted definition of so-called normal vision. Hence any object which is barely visible through this portion of the gradient filter is assigned a value of unity in visibility.

A scale value of "2" for example, indicates that the visibility of the object of regard under the same test conditions, is equivalent to that of the standard test object when the latter subtends a visual angle of two minutes. Thus we express visibility in terms of threshold size of object as does the ophthalmologist.

The scale of visibility of the L-M Visibility Meter is based merely upon the assumption of a series of test-objects of similar form but varying in size from the smallest visible to one equivalent in visual size to the area of the fovea. Several objects from this series are illustrated in Fig. 11. Obviously, the visibility of any object, viewed under any conditions by any observer may be expressed as equivalent to that of one of the series of standard black test-objects viewed against a white background under certain specified conditions. In other words, the standard test-object may be regarded as a "common denominator" for expressing the relative visibilities of an infinite variety of objects which vary in size, shape, contrast and brightness. For example, it will be noted from Fig. 11 that the visibility of 3-point, 6-point, 12-point, and 18-point type (black on white paper) is equivalent to that of standard test-objects which subtend visual angles of 1.10, 2.64, 5.66 and 8.67 minutes, respectively, under the same level of illumination.

In general, it is well known that the effectiveness of size, as an aid to seeing, follows a law of diminishing returns. This is clearly recognized in the science of ophthalmology. For example, if a patient can barely recognize objects whose critical details subtend a visual angle of two minutes rather than the normal value of one minute, he is considered to possess a visual efficiency of 84% rather than 50% which might be supposed upon a basis of visual acuity. Similarly the numerical values of visibility obtained with the Visibility Meter, expressed in terms of the size of a standard test-object of equivalent visibility, are not directly proportional to the *actual* or *effective* visibilty. However, these values may be used as a basis for deriving a scale of visibility for which the individual values are directly comparable. We have evolved such a scale[11] and have termed it as a scale of "Per Cent Maximal Visibility." Thus for the first time it appears to be possible to appraise the increase in visibility produced by any aid to seeing upon a percentage basis, the upper limit of which may be described as that corresponding to maximal visibility.

The term "maximal visibility" is used herein to designate an extremely high degree of visibility which serves as a practical limit. In general, the maximum of visibility which we have adopted

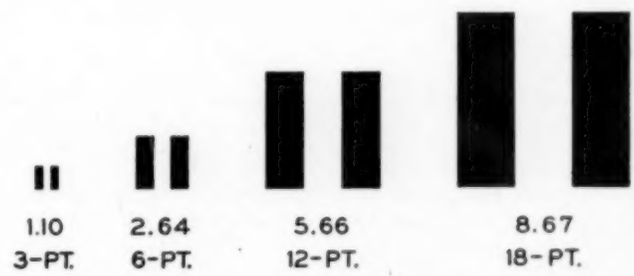


Fig. 11.
Illustrating the relative visibility of various sizes of type in terms of our standard parallel bar test-object. These objects should be viewed from a distance of 10 feet in order to subtend the designated visual angles

may be appreciated from the fact that it represents a degree of visibility equivalent to that of objects indefinitely large in size and of maximal contrast under a brightness-level of 10 foot-lamberts. In ophthalmology, a person who could not recognize such an object would be designated as "blind."

It is now of interest to determine the per cent of maximal visibility which may be obtained by augmenting various controllable factors. For example, we may ascertain the actual increase in visibility obtained with various sizes of type viewed under a constant level of illumination. These data are presented in Fig. 12 and pertain to

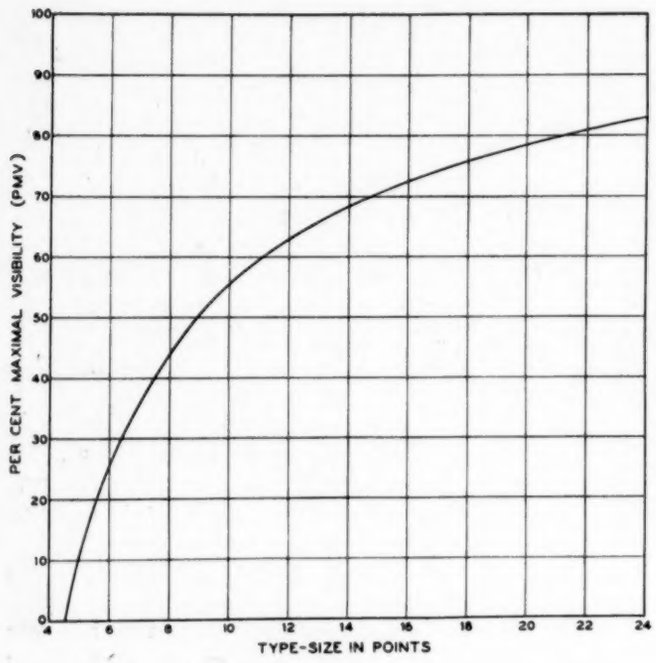


Fig. 12.
The per cent of maximal visibility (PMV) obtained with various sizes of well-printed Bodoni Book Monotype under an illumination of 10 footcandles. These data were determined for normal vision and a reading-distance of 14 inches

excellent printing with black ink upon white paper as viewed by persons possessing normal or near-normal vision. The illumination in all cases was 10 footcandles. It will be noted, for example, that about 60% of maximal visibility is obtainable with 12-point type under an illumination of 10 footcandles. Type of this size is the largest commonly used in the printing of books. These data have many practical usages. For example, in those situations in which books printed in 8-, 10-, or 12-point type are being considered for use in schools, the relative visual advantage of the larger sizes are definitely known and hence may be weighed against costs.

The data of Fig. 13 show the increase in the visibility of 8-point type, expressed as a percentage of maximal visibility, for various levels of illumination from one to 100 footcandles, respectively. It will be noted that about 90% of our standard of maximal visibility is obtained when the illumination on the 8-point type is 100 footcandles. This conclusion is based upon the assumption of so-called normal vision in obtaining these data. Obviously a person with less than normal vision would obtain a smaller percentage of maximal visibility under similar conditions. Thus it appears from these data that 8-point type can be brought to a high degree of visibility through the utilization of levels of illumination which are practically and economically obtainable. However, type of this size is often read under 5 footcandles or less. If 5 footcandles is assumed as an example, it will be noted than less than 30% of the possible degree of visibility is obtained under this level of illumination. Furthermore, the data of Fig. 13 pertain to excellent printing with black ink on a good grade of white book paper. If instead of the latter ordinary newsprint is used, the degree of visibility achieved under 5 footcandles is commonly less than 10% of the maximal value.[1]

Not infrequently the assertion is made that 5 or 10 or 15 footcandles are adequate for reading. If we ignore, for the moment, the differences in the visibilities of various kinds of printed matter, it is obvious from Fig. 13 that the foregoing statement is highly arbitrary. Certainly it is not based upon quantitative considerations of visibility. Under 15 footcandles persons with normal vision obtain only about 50% of maximal visibility for well-printed 8-point type. This value may be raised to 70% by increasing the illumination to 30 footcandles. Newspapers are commonly printed with 7-point type.

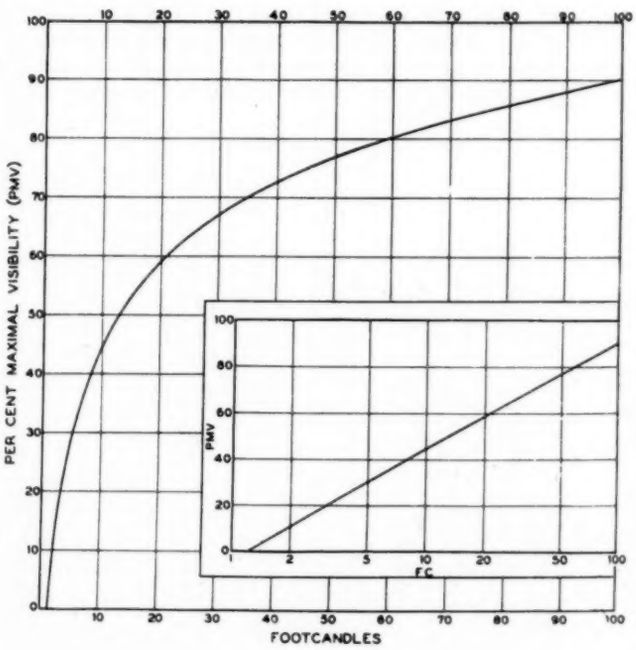


Fig. 13.
The per cent of maximal visibility (PMV) obtained with well-printed 8-point Bodoni Book Monotype under various levels of illumination. The insert shows the same relationship with the footcandle values plotted logarithmically

A diagrammatic view of any aid to seeing is shown in Fig. 14. The arrow represents any aid to seeing, of which light and lighting are universally available and controllable aids. One may change the position of the arrow by turning the knob *K*. By turning this knob arbitrarily, and thus without consideration of the relation of monetary costs to human welfare, we may demonstrate how some dogmatic conclusions are arrived

at. Any setting of the arrow actually represents a compromise between costs and benefits from any specific aid to seeing. In the lowest position the specific aid is utilized minimally. It may be illumination for barely seeing; or it may be the smallest type-size that can be seen under given conditions. It may be any (or a combination) of all the various controllable aids to seeing. No one can evaluate human benefits in dollars as are lamps, fixtures and kilowatt-hours. Still a variety of naive conclusions are continually expressed which are obviously based upon the incomplete economics of lighting. What we can *afford* may change with time and knowledge, as well as with economic status. Besides, many of these conclusions are based upon the false assumption that 100 footcandles cost 100 times as much as one footcandle. No consideration is given to the desirability of obtaining much of the light by directed or localized light combined with a lesser but desirable level of general illumination. In this manner both light and lighting are properly combined from the viewpoint of seeing for efficiency and welfare of human beings.

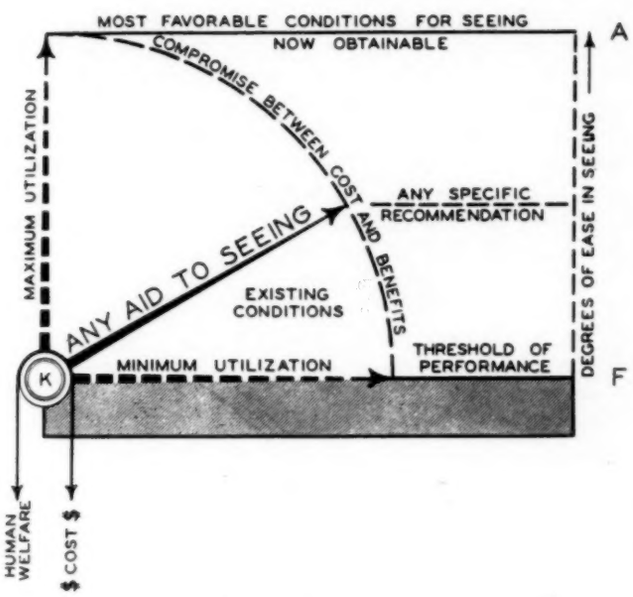


Fig. 14.
The degree of utilization of any aid to seeing (type-size, footcandles, lighting, etc.) is represented by the position of the arrow which also represents a degree of ease of seeing. Excepting for the condition of maximum utilization of any aid to seeing, any position represents a compromise between monetary cost and human benefits or welfare

Returning to Fig. 14, let us consider recommendations regarding desirable conditions for seeing. The average type used for the text-matter in newspapers is approximately 7-point in size. It is obvious that 10-point and 12-point sizes which are used in many books are more desirable. Recommendations should be above the average until practice has attained the desirable standard. This is equally true of type-sizes and footcandles as well as of other aids to seeing. Footcandle recommendations were almost entirely arbitrary for many years. The science of seeing has evaluated them as steps toward ideals which have been revealed with some degree of clarity in recent years. Thus there can be no sound opposition to a gradual elevation of footcandle recommendations if lighting practice improves accordingly. The person who asserts that five footcandles are enough for reading should know that the reading-matter he has in mind can be read under a fraction of a footcandle. Why did he "turn the knob" in Fig. 14 from a fraction of a footcandle to 5 footcandles? Why does he leave the arrow there? Whether he realizes it or not, he is compromising between costs and benefits but he has no right to imply that he has done this by sound analysis and with complete knowledge. Neither he nor anyone else can fix this location irrevocably. Would he still say that five footcandles are enough for reading or for study in schools, if the luminous efficiencies of light-sources were suddenly increased ten-fold? His ideas of "enough" would likely change if light-sources emitting 20 lumens per watt were replaced by new ones emitting 200 lumens per watt. Admittedly monetary cost must be considered, but economics are too indefinite and flexible and benefits cannot be evaluated adequately to establish a recommendation of footcandles definitely and finally.

These glimpses of new approaches toward an understanding of seeing as contrasted with the narrower concepts of vision reveal that a science of seeing[1] is in the making. It is gratifying to note the increasing interest of ophthalmologists and physicians in these new vistas and in the opportunities which they afford for still greater service in the realms of human efficiency, comfort and welfare.

References:

1. The Science of Seeing, MATTHEW LUCKIESH and FRANK K. MOSS, D. Van Nostrand Company, New York, 1937.

2. Light and Work, M. LUCKIESH, D. Van Nostrand Company, New York, 1924.

3. Fatigue of Convergence Induced by Reading as a Function of Illumination Intensity, M. LUCKIESH and FRANK K. MOSS, *Am. J. Ophthal.*, 18, 1935, 319.

4. A Correlation Between Illumination Intensity and Nervous Muscular Tension Resulting From Visual Effort, M. LUCKIESH and FRANK K. MOSS, *J. Exper. Psych.*, 16, 1933, 540.

5. The Effect of Visual Effort Upon the Heart-Rate, M. LUCKIESH and FRANK K. MOSS, *J. Gen. Psychol.*, 13, 1935, 131.

6. On the Act of Blinking, E. PONDER and W. P. KENNEDY, *Quart. J. Exper. Physiol.*, 1927-1928, 18, 89.

7. The Eyelid Reflex As a Criterion of Ocular Fatigue, M. LUCKIESH and FRANK K. MOSS, *J. Exper. Psychol.*, 20, 1937, 589.

8. Effects of Leading on Readability, M. LUCKIESH and FRANK K. MOSS, *J. App. Psychology*, 22, 1938, 140.

9. Visibility and Readability of Print on White and Tinted Papers, M. LUCKIESH and FRANK K. MOSS, *Sight-Saving Review*, 8, 1938, 123.

10. Visibility — Its Measurement and Significance in Seeing, M. LUCKIESH and FRANK K. MOSS, *J. Frank. Inst.*, 220, 1935, 431.

11. Thresholds and Supra-Thresholds of Seeing, M. LUCKIESH and FRANK K. MOSS, (Accepted for publication in the Transactions of the Illuminating Engineering Society).

Intra-Articular Damage

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IN DISCUSSING intra-articular damage in a generalized fashion, we shall attempt to offer a few suggestions concerning what is often spoken of as "the handling of an industrial case."*

Patients suffering from intra-articular lesions frequently have definite and fixed ideas. They assume, for example, that since a joint has been damaged it is prone to become "stiff," and that operative interference will only make a bad matter worse. (In business, the customer is always right.) Fortunately, in industrial surgery, this is not necessarily true.

In fractures of the shafts of the long bones, there is, as a rule, an immediate total disability. Joints, on the contrary, may continue to function under most adverse circumstances. Patients who are not completely incapacitated (and do not require emergency treatment) are prone to ask innumerable perplexing questions. These must be answered to their satisfaction. To do this, we must "speak the patient's own language." This also holds true in giving testimony before referees and juries, or in submitting reports to be read by claim adjustors.

We all realize that in joint injuries, many intricate biochemical phenomena occur, which we ourselves do not fully understand. However, in trying to simplify matters for others, we often form new mental concepts for ourselves. Mechanical comparisons are helpful, now that the automobile has given most people a conversational acquaintance with mechanics.

Practically all patients of average intelligence appreciate knowing what is wrong with them. Their whole-hearted cooperation is most essential; particularly in the treatment of what might be called "a working part."

We try to impress upon our patients:

(a) That their joints will be no stronger than the muscles governing them.

(b) That muscular power can be developed in only *one* way, i.e., through *voluntary* exercise.

(c) That a distended joint is analagous to a "hot box" into which nature has poured an overabundance of "lubricating oil."

(d) That an increase in the joint fluid is an indication that something is "wrong" within the joint cavity.

After the effusion has subsided, usually at the end of the second week, we urge our patients to "try-out" the joint. "Put it to its full use." If there is a recurrence of the swelling, we may then be quite certain something is radically wrong. Approached in this manner, practically all our patients have been willing to cooperate in what we have come to call a "try-out test." These tests have proved most helpful in establishing a final diagnosis. More than this, we believe they can be relied upon as an indication for operative interference. To quote the late Sir Robert Jones, "the indication for operation is *repeated* attacks with effusion."

Many apparently intelligent individuals follow fads and fancies. This fact does not imply, however, that they are not anxious to obtain information concerning the pathological factors responsible for their symptoms.

Pathology

LET US consider a few of the more important lesions:

1. *Intra-articular fractures.* The underlying bone is close proximity to an articulation, is of a *soft, cancellus* nature and apparently designed to undergo *"stress* and *strain."* Rarely, if ever, do intra-articular fractures fail to unite. On the contrary, our problem is one of preventing an over-production of bone. Bony over-growths arise from three sources:

(a) From the joint "housing" or capsule;

(b) From the adjacent bone;

(c) From within the surrounding musculature, namely a myositis ossificans.

In our opinion, repeated forceful manipulations of recently traumatized joints tend to set up an over-activity of the connective tissue cells which later become converted into cartilage and finally, into bone. Therefore, early operations undertaken for the removal of bony blocks, would seem to be contra-indicated. In our opinion, the time to operate is after there are no roentgenological signs of cellular activity. This may mean that an operation should be postponed for many months, perhaps a year, following the date of injury.

We hesitate to suggest that intensive heat may have a similar undesirable effect upon recently traumatized articulations. Baking is almost universally prescribed and said to promote synovial absorption. In a fully distended knee (containing as much as 185 cc. of fluid) we have repeatedly measured the circumference two or three hours following the local application of heat, only to find that the joint was, if anything, a trifle larger than it was prior to the beginning of treatment.

We have noted that bony over-productions rarely occur at the point where the fracture line enters the joint cavity. On the contrary, a definite niche often marks the site of a bony lesion. In intra-articular fractures, overly distended joints tend to decompress themselves through the fracture line. As has been said, intra-articular fractures unite favorably and we, therefore, hesitate to suggest that the presence of synovial fluid may be a factor in the prevention of bony proliferations.

Using utmost care not to damage the surrounding parts, we have resected a shattered head of a radius, only to find that, two months later, the entire anti-cubital space was completely filled with new bone. Aside from any know factors, there are certain individuals who apparently have a definite tendency toward the over-production of bone. In our experience this has been particularly

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* Presented at the Twenty-Third Annual Meeting of the American Association of Industrial Physicians and Surgeons, and Midwest Conference on Occupational Diseases, Chicago, June 6-9, 1938.

evident in young individuals, but rarely encountered in those over 50.

II. In considering injuries to the *fibro-cartilaginous cushions*, our attention is at once centered upon the knee. It should be remembered, however, that (a) the triangular fibro-cartilage of the wrist, (b) the acromio-clavicular cartilage, (c) the stylo-mandibular cartilage, and (d) the intervertebral disc, are also occasionally damaged.

In the knee, the internal meniscus is injured 50 or more times more frequently than the external cartilage. It is firmly attached to the internal lateral ligament and probably drawn out of position by this structure. The condition can only be produced by a twisting violence. Once the cartilage is displaced, it is probably crushed between the bone ends, this producing either a transverse or a longitudinal fracture.

We explain to our patients, that cartilage is not usually visualized in x-ray. We insist, however, upon having *carefully prepared comparative roentgenograms made*, in order to rule out any associated bony lesions. Following this, we explain to the patient that there are no blood vessels within the fibro-cartilage and, therefore, a satisfactory union or repair cannot be expected. Many patients treated conservatively for months or even years, when they come to realize their true condition, seriously consider the only possible curative procedure, namely, operation.

In industrial cases we are guided, to a considerable degree, by the patient's occupation. Some are able to carry on. We discourage such patients into believing that prolonged expensive treatments will prove beneficial. In those who are doing heavy work and are continually or repeatedly disabled, we strongly urge operation. Often we have to explain that the relief obtained from wearing a plaster of Paris cast is only temporary, and that the wearing of casts or the use of braces seriously interferes with the function of the muscles governing the damaged joint. Muscular atrophy following disuse may often be detected as early as the second week. In fact many patients themselves have observed a marked diminution in the circumference of the thigh on the affected side.

III. Let us consider the *smooth, glistening articular cartilage*. Undoubtedly it is affected by most joint injuries. However, so far as we know, trivial lesions do not produce any permanent condition. We question, therefore, whether such cases should be dignified with the diagnosis of "traumatic arthritis." Many of the more severe lesions of the articular cartilage, are now classified as "arthritis dessicans." This condition may be analagous to "a pitting process." Sharply punched-out portions of articular cartilage (perhaps as a result of an accident) are torn or rubbed away from their moorings. Undoubtedly these loose bodies grind about within the joint. They actually live in the synovial fluid and there is little or no likelihood that they will become disintegrated or absorbed. Eventually, then, they must be removed. We are not prepared to state whether, following a careful resection of the overhanging edges of the areas from which these loose bodies were derived, there will occur some further cartilaginous desiccations.

IV. The "joint lining," or *synovial membrane*, is in reality a highly specialized connective tissue. It becomes thickened from injury or infection and folds upon itself until villi are produced. Perhaps as a result of an injury, some of these villi may become detached producing rice bodies. Overgrowths of the synovial membrane in the nature of a pannus may eventually spread across the articular cartilage or completely cover over the fibro-cartilaginous cushions. This, in our opinion, is more apt to occur in older patients suffering from hypertrophic changes. Their joints squeak, as it were, during flexion and extension. This sound is quite different from the clicking caused by large loose bodies. A resection of the hypertrophied synovial membrane may be indicated when a joint is opened for some other intra-articular lesion. Animal experimentation has shown that an early reformation of the resected joint lining is to be expected. Therefore we do not care to make a definite statement concerning the advisability of a synovectomy as a primary procedure. In most of our industrial cases we have not recommended it.

Pads of fat are closely associated with, and perhaps a part of, the synovial membrane. Those in the anterior joint space of the knee frequently become "pinched." The margins become definitely thickened and hardened, and show evidence of recent hemorrhage. We strongly recommend their resection. The body weight apparently has no bearing, since we have repeatedly encountered traumatized fat pads in those underweight. We recall two cases, previously operated upon for the removal of a fractured fibro-cartilage. It was only after removing the fat pads that we were able to return these men to their usual occupations.

V. Finally, we come to a most trying problem, the *aggravation* of *pre-existing conditions*. In the roentgenograms of most of these cases, an extensive hypertrophic osteoarthritis can be clearly demonstrated. In perhaps a half dozen instances we have seen what appeared to be recent fractures of pre-existing bony spurs. The joint surfaces are, as it were, "squashed out" or "flattened," but, in our experience, chips are not likely to fly away in the same manner as they do from the edge of a flattened hammer.

It is difficult for us to believe that a marked aggravation of a pre-existing condition would not produce a joint effusion. If so, when the effusion has subsided, is it not fair to believe that the period of aggravation has terminated? We are inclined to answer affirmatively.

Unfortunately, in back injuries we are unable to detect an effusian. We have, therefore, to rely upon (a) muscular spasm, (b) definite *point* tenderness and (c) limited motion. In cases of long standing, if there has been an aggravation due to a recent injury, it is our opinion that x-rays made at intervals of three or four months covering a period of one year, should show progressive bony changes. The absence of such changes (excepting those which might occur in the course of disease) leads us to believe that the effects of any aggravation have completely subsided.

Reasoning along these lines, we have been able to convince several patients that they were no longer suffering from the effects of a recent injury. Of course, it would be useless to attempt to reason with willful malingerers, or to try to convince those suffering from hysteria that they were over the effects of a recent or remote accident.

In our opinion, many of the difficulties encountered in treating joint injuries, may be attributed to an alteration in the local circulation (which in all probability influences the local chemistry both within and about the joint). The early emptying of an over-distended joint by means of aspiration, is frequently indicated and will relieve, at least to a certain degree, the circulatory embarrassment.

In connection with aspirations, there is a diagnostic point which may prove helpful in fixing the date of an "alleged injury." The fluid from a recently traumatized joint is almost invariably bloody. On about the tenth to the twelfth day, or later, it regains its normal macroscopic appearance. (We believe, however, these findings require further confirmation.)

New York Central Surgeons

THE fifteenth Annual Meeting of the Association of Surgeons of the New York Central System will be held at the Biltmore, New York City, on Monday, Tuesday and Wednesday, October 24, 25 and 26, 1938.

The Program Committee, under the chairmanship of DR. B. L. COLEY, Chief Surgeon at New York City, has arranged a splendid program, the details of which follow.

This is again a three-day meeting arranged with a view to presenting a satisfactory program, while affording time for recreation and sight-seeing in New York City. Due to the fact that there are so many points of interest in New York, no formal program of ladies entertainment has been planned.

The annual complimentary dinner for the attending surgeons and their wives will be held in the Ballroom on the nineteenth floor of the hotel on Tuesday evening. A reception will be held from 6:30 to 7:00 P.M., in the Fountain Court adjoining the Ballroom, to which all members and their ladies are invited. This will give the members an opportunity to meet MR. O. G. BROWNE, Mr. Whiting's successor as General Claims Attorney.

MONDAY — October 24

Scientific Session

9:30 to 12:00

9:30 A.M.—General Meeting, Music Room, 1st Floor.

President's Address — HARLOW G. FARMER, M.D., Watertown, New York.

Fracture Symposium

"Fractures of the Elbow"—PHILIP D. WILSON, M.D., New York.

"Fractures of the Lower Leg and Ankle"—CLAY R. MURRAY, M.D., New York.

"Fractures of the Femoral Shaft"—ROBERT H. KENNEDY, M.D., New York.

"The Role of the Railroad Surgeon in Improving the Standards of the Treatment of Fractures"—FREDERIC W. BANCROFT, M.D., New York.

Discussion opened by BRADLEY L. COLEY, M.D., New York, and HAROLD H. BAKER, M.D., Rochester, New York.

Afternoon Session

1:30 P.M.—Golf Tournament — Siwanoy Country Club, Pondfield Road, Bronxville, New York.

2:00 P.M.—Oculists Meeting.

Clinical Session

New York Eye and Ear Infirmary, 13th Street and Second Avenue, New York.

"Disease of the Eye"—CLYDE E. MCDANNALD, M.D., and TRUMAN L. BOYES, M.D., New York.

TUESDAY — October 25

Clinical Session

9:30 to 12:00

9:30 A.M.—Bellevue Hospital, 415 East 26th Street, New York. (Program to be announced at meeting)

Oculists Meeting, Rooms 101 and 103, 1st Floor, the Biltmore.

Round Table Discussion—"Ocular Diseases."

"Ocular Motor Anomalies"—JAMES W. WHITE, M.D., New York.

Scientific Session

2:00 P.M.—"Repair of Supraspinatus Tendon"—JOHN E. SULLIVAN, M.D., New York.

Discussion opened by ARTHUR M. DICKINSON, M.D., Albany, New York.

"Nucleus Pulposus: Transportation of Back Injuries" — BURTON P. STOCKEY, M.D., New York.

Discussion opened by LEONARD A. ENSMINGER, M.D., Indianapolis, Indiana.

"Treatment of Traumatic Wounds with Zinc Peroxide" — FRANK L. MELENEY, M.D., New York.

Discussion opened by BROOKS W. MCCUEN, M. D., Syracuse, New York.

"Sulfanilamide"—GORDON MYERS, M.D., Detroit, Michigan.

Discussion opened by CHRISTOPHER D'AMANDA, M.D., Rochester, New York.

Business Session

Reports of Committees

New Business

Election of Officers

6:30 to 7:00

6:30 P.M.—Get Acquainted Hour. General Reception for members and their ladies in the Fountain Court, adjacent to Ballroom, nineteenth floor.

7:00 P.M.—Annual Dinner in the Ballroom on the nineteenth floor MR. O. G. BROWNE, General Claims Attorney, Toastmaster. MR. R. E. DOUGHERTY, Vice-President, Improvements and Developments, New York.

MR. J. ARONSON, Vice-President, Law, New York.

Distribution of golf prizes.

Music and entertainment.

WEDNESDAY — October 26

Clinical Session

9:30 to 12:00

9:30 A.M.—Presbyterian Hospital, 622 W. 168th St., New York. (Program to be announced at meeting.)

2:00 P.M.—Oculists Clinic—Institute of Ophthalmology of the Presbyterian Hospital, Fort Washington Avenue and 165th Street, New York.

"Diseases of the Eye"—JOHN H. DUNNINGTON, M.D., New York.

Industrial Medicine

Reg. U. S. Pat. Off.

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A. A. I. P. & S. Committees

THAT part of any profession which is engaged in a special line of work has the right and the duty to develop that special line to the fullest possible extent of all the present and potential circumstances to which its brains and ability are applied. In law, for example — as between plaintiff and defendant in the civil practice, and prosecution and defense in the criminal practice — the lawyer who is on either side is, for the purposes of his work, wholly and completely on the side of his choice, intense in his preparation, earnest in his advocacy. In medicine there is no such opposition of attitudes — because the patient is always the patient. In medicine the special circumstances which differentiate one field of practice from another are inherent in the conditions under which the patient comes to the Doctor. He comes from either an industrial source or a non-industrial source. From the former, he arrives with the employer and the employer's liability behind him. He comes from the background of the triad which, as DR. BARTLE says (p. 631): "must be respected in all of our consideration —the industry, the worker, and the Doctor."

The Doctor, therefore, owes it to all three to be thorough in his preparation, qualified in his experience, earnest in his treatment, and capable in his judgment. And he owes it to all three to cooperate whole-heartedly in the efforts of his Association to require, and to set up and maintain standards for the necessary preparation, experience, earnestness and capability in the field where the doctor-patient relation *is* the triad as against the duet. That relation is a *special* field, and it does have its own *special* requirements.

The work of the American Association of Industrial Physicians and Surgeons in this respect — particularly as indicated by its new Committees and the significances to be inferred from their purposes as expressed by their names — is emphatically something with which to cooperate along these lines. President C. D. SELBY has appointed the A. A. I. P. & S. Committees for this year as follows — the Chairman in each instance being the first named. All of them have an important place in the Association's growth and development; some of them have a very significant relation to the forward progress of industrial medicine in general:

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Cooperation of Railroad Surgeons

W. J. LANCASTER, M.D.,
Wilmington, North Carolina

FOR the sake of convenience I have sub-divided this subject under the following heads: Qualification for a Railroad Surgeon; Brief Resume of Railroad History; The Duty a Railroad Surgeon Owes to His Company; The Duty a Railroad Surgeon Owes to His Community; The Medicolegal; and The Assistance that the Railroad Surgeon Can Give Both to the Railroad and the Public.*

Qualification

HE MUST be a temperate man, and in using that word temperate I do not confine the meaning to alcohol. It is just as necessary that he be temperate in regard to eating, sleeping, exercise and habits in general.

He must be qualified in his profession, and this qualification takes in more than the mere knowledge that he gains in medical schools, hospitals and from books. The right type of man must be level-headed, aggressive, sincere, love his work, and in addition to this, give his time, thought and attention to his work.

He must keep his own self respect and in doing this he is in position to demand respect from others.

He should make his reports to the central office so that the office will have a word picture when the report is read. You know the central office frequently has to pass upon questions on the report of the surgeon alone with no other history. Please make that report as concise and to the point as possible, but at the same time cover the subject from all angles.

A railroad surgeon should be an outstanding man in his community, a leader in civic affairs, as well as having a high professional standing. His honor and integrity should be above reproach and his veracity unquestionable.

He must keep himself in good physical condition. This is a duty he owes himself, his family, the railroad company and the public at large.

He should be fair, impartial and thorough in his reports of accidents. No railroad company wishes any of its personnel to misrepresent facts for them or misrepresent conditions to them.

Brief Resume

IN 1802 Richard Trevethick took out a patent for a wheeled engine which should run on rails by its own power. In 1814 George Stephenson built a locomotive and put it in operation near Killingsworth, England, demonstrating that a smooth-wheeled engine on smooth rails could draw loads up hill. The Manchester and Liverpool Railway was opened for passenger and freight traffic in 1830. These happenings all occurred in England. The first railway built in the United States, especially for the purpose of steam traffic, was the one begun in South Carolina in 1830.

The principles that George Stephenson advanced in his engine, The Rocket, were sound then and are sound today. If it had not been for the railroads blazing the trail, that vast section of the United States west of the Mississippi would still be a wilderness. If it had not been for railroads all the important industrial centers would still be p.airies, because railroad transportation collects and hauls coal and oil to the factories, carries raw materials to the factories, and transports finished products to every hamlet in every State of the Union. Do you know that the railroads partake of the results of research of almost every producer of 70,000 different items of materials, supplies and equipment, which they buy and use in the manufacturing of their products of service? Do you know that Thomas Edison, the electrical wizard, got his start in the railroad business, and that Walter Chrysler and Vinson Bendix, of automobile fame, learned the mechanical trade in railroad shops?

Railroads do not manufacture articles to sell to consumers; they buy things and combine them to produce a service which they sell. The approach of railroads is different from that of a business which makes and sells products, and which may in fact make a considerable part of this year's profits by outmoding the products of last year in the hands of the consumer. The problem is totally different, too, in that everything now introduced to a railroad must fit in and work right along with what is already there. The automatic coupler, for example, could not be introduced in a practical way until it was so developed that it would work interchangeably with any one of the several sorts of hand operated couplers then in use. Everything on a railroad is part of one great organization of men and machines operated for the production of a service. I use this purely as an illustration of the word cooperation. Do you know that three years ago six railroad companies, two universities, and 10 supply houses, alone had more than 1000 men engaged in research on railroad problems, and since that time the number has increased? Mangement, labor, and the public, all three, have a vital interest in railroads, and all three should work together for the common good of all concerned.

In the history of the building of railroads the surgeon was just as necessary as the construction engineer or any other official. Without the advice of the surgeon on sanitation, epidemics and keeping the health of the forces used in construction work in good condition, no railroad could have been built. In support of this statement I call your attention to the Panama Canal. As you know, this undertaking had been tried before by at least two different countries and they failed, because of the sanitation and health conditions, until two great men, one an engineer and one a physician, together worked out a plan whereby the health and sanitation of the workers were made safe. As a result of the work of Gorgas and Goethels the Panama Canal was made a reality. This same type of cooperation between engineers and doctors made possible the building of all railroads.

I would like you to make a mental picture of the early railroads, their tracks and equipment, and note today the wonderful locomotives, the air conditioned coaches, the rock ballasted roadbeds, the automatic block signals, the pleasure and comfort of sleeping, the splendid meals in dining cars, the high speed of both passenger and freight trains, and to take into consideration the number of trains running daily with all the obstacles that are placed upon the railroads, such as weather conditions, road crossings, etc. As you know, it is now not a railroad crossing any more, but an automobile crossing over railroad property. With these obstacles in mind, it is really remarkable how close to schedule time the trains run. At the same time, I wish you to have a mental picture of the medical profession in 1812, and see what progress they have made from the old days when the barber was the surgeon, when no antisepsis or asepsis was used, to the present

*Read before the Annual Meeting of the Florida Railway Surgeons' Assn., St. Petersburg, April 5, 1937; published in the *Journal of the Florida Medical Association*, August, 1938.

day set-up of your Mayo Clinics, your Johns Hopkins Clinics, your Crile Clinics, your Lahey Clinics, your Tulane Clinics, and others too numerous to mention. Cooperation has been necessary in our own profession, both within its own ranks, with the public, and with corporations like railroads who have helped to make the progress possible. Railroads carry and safely transport physicians to distant medical centers for special study and work. They also carry safely and with every convenience transport your patients to centers that are especially equipped to handle certain types of cases.

Duty to His Company

TIMES and customs have changed. We have gone forward and in going forward the railroad surgeon has had to keep pace with the forward movement. I am outlining a few points that are pertinent to these changed conditions.

The treatment a railroad surgeon renders an injured patient is public property and his method and type of treatment is frequently aired in court, so that a railroad surgeon in rendering correct, efficient and courteous treatment to an injured person not only serves the railroad but also usually makes a friend of the patient and the patient's family, and thus opens up a legitimate avenue for future private work.

In addition to being qualified from a professional standpoint he must be prompt and comprehensive in his reports, give a fair, accurate and thorough account of his physical findings, including those conditions which in his opinion were caused by the accident and also all abnormal conditions that are found which, while in his opinion they have no connection with the accident, the knowledge of them will be of inestimable value to the railroad company at a later date.

The railroad surgeon should stay strictly in his own profession and properly treat the injured. He should not try to furnish the necessary medical and surgical treatment to an injured person and at the same time act in the capacity of a claims adjuster, because if he does assume this dual role, both he and the company lose in the long run, as the patient loses confidence in the surgeon and that hurts both the surgeon and the railroad company. If the surgeon does assume this dual role while he is still attending the patient professionally, he frequently loses his professional equilibrium, his mode of treatment sometimes will be changed, due to the reaction produced on the patient when the surgeon while treating him professionally attempts to inject the settlement of the case into his treatment. After he has finished treatment it is his duty, if and when called upon by the Claims Department, to assist

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of

INDUSTRIAL PHYSICIANS and SURGEONS

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Central States Society of Industrial Medicine & Surgery

Association for the Advancement of Industrial Medicine & Surgery

THE object of this Association shall be to foster the study and discussion of the problems peculiar to the practice of industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes and results of the medical care of employees, and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for the mutual advancement in the practice of their profession.

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them in any way that he can, and this, at this time includes rendering them assistance in the settlement of the case, but never under any circumstances trying to handle the case professionally, and at the same time handling the claim end also.

The surgeon should be frank and fair in talking to the patient. However, he should carefully weigh his words in talking to an injured person as the patient frequently is apt to interpret the expressions of a railroad surgeon to suit his own situation and condition. Remember, that an injured patient frequently will have a wonderful memory of what you said when you first saw him and this sometimes in the future is embarrassing to doctors.

Don't be either too optimistic, or too pessimistic, as either extreme places your company in an embarrassing position. Examine your patient thoroughly, completely and conscientiously. This type of examination will be beneficial to the patient, your company and yourself. If you are too optimistic at first and find out a little later that some serious complications develop in your patient, you destroy the confidence of your patient and bring criticism on your company and on yourself. If you are too pessimistic about the future of the patient, you create an unnecessary hazard for the company and frequently sow seeds in a fertile ground that are expensive to the company in court later on. In addition to these facts you produce disastrous results on the patient himself, not only with regard to your immediate treatment, but also to his future well being. Psychologically, this will play an important factor. Be thoughtful, fair, frank, impartial and conservative in expressing your opinions. Above all, never fail to acquaint the company with expected bad results as well as expected good results in all cases.

Duty to His Community

FIRST: That he keep his physical and moral condition up to the standard at all times.

Second: That he be up-to-date, especially with reference to his profession, and also up-to-date in civic affairs for the good of his community.

Third: He must be careful, considerate, broadminded, and primarily devoted to his profession.

Fourth: Such a man will necessarily be civic minded.

Fifth: To conduct himself both personally and professionally so that he will be loved and respected and live so that his honesty, integrity, veracity and ability will not be questioned.

The Medicolegal Aspect

IN TALKING about the medicolegal side of the railroad surgeon I wish to emphasize the following:

You have thoroughly and completely examined the patient, unless, of course, you are going to testify purely as an expert and testify only on hypothetical questions.

In advance of going on the stand, you should have several conferences with the railroad attorneys and go over in detail the claims made by the plaintiff, comparing the alleged injuries with the facts found at your examination. Compare also the present condition of the plaintiff with the type of injuries received and frankly discuss your views with the attorney. Above all, give the attorney the dark side as well as the bright side of the situation from a medical standpoint, as it is much better for both to thoroughly understand the situation before you get to the court room. Thoroughly prepare yourself about the condition you found at the time of the accident, the present condition of the patient (if you are allowed to examine the patient at this time), and the ultimate result that more than likely will follow a severe injury. Do not trust to memory; but put down all your findings as you examine the case and do not wait until you get back to the office for this important feature. Also put down the date of examination, where made, and who was present at the time of examination. Again let me emphasize the importance of a thorough and detailed examination, noting all conditions found that are abnormal whether relating to the accident or not. Also remember to be very careful in expressing your views of the case, as unfortunately, due to the stress of excitement immediately following an injury, the patient and the attending public at any scene of an accident will be very apt to distort what was said by the physician.

On the stand, never lose your self control. If you have prepared yourself beforehand and have consulted frequently about the case with the attorneys who engaged your services, you need have no fear of proper examination by the other attorneys. If your examination is improper the attorney who engaged you and the judge will handle that part for you. Use as few technical phrases as possible; use a language that the judge, jury and attorneys will understand and you will have less chance of being severely cross-examined by the opposing counsel. Answer the questions asked you as much to the point as possible without including in your answers subjects not relating to the questions asked. Finally remember that you are a doctor and not a lawyer, that the company and the Court expect you to give an honest and truthful reply to the medical questions asked. Then it is up to the legal profession to apply the medical knowledge that you have given

The Industrial Doctor

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STATE OF WISCONSIN } ss.
COUNTY OF ROCK }

Before me, a Notary Public, in and for the State and county aforesaid, personally appeared C. F. Karstaedt, who, having been duly sworn according to law, deposes and says that he is the Secretary-Treasurer of the Industrial Medicine Publishing Company and that the following is, to the best of his knowledge and belief, a true statement of the ownership, management, etc., of the aforesaid publication for the date shown in the above caption, required by the Act of August 24, 1912, as amended by the Act of March 3, 1933, embodied in section 537. Postal Laws and Regulations, printed on the reverse of this form, to wit:

1. That the names and addresses of the publisher and editor are: Publisher: Industrial Medicine Pub. Co., Beloit, Wisconsin. Editor: A. D. Cloud, 540 N. Michigan Ave., Chicago, Illinois.

2. That the owners are: A. D. Cloud, 533 Madison St., Glencoe, Illinois; A. G. Park, 347 Jackson Blvd., Glencoe, Illinois; R. F. Collins, 1138 Highland Ave., Beloit, Wisconsin; C. F. Karstaedt, 820 Milwaukee Road, Beloit, Wisconsin.

3. That the known bondholders, mortgagees, and other security holders owning or holding 1 per cent or more of total amount of bonds, mortgages, or other securities are: There are none.

4. That the two paragraphs next above, giving the names of the owners, stockholders, and security holders, if any, contain not only the list of stockholders and security holders appears upon the books of the company as trustee or in any other fiduciary relation, the name of the person or corporation for whom such trustee is acting, is given; also that the said two paragraphs contain statements embracing affiant's full knowledge and belief as to the circumstances and conditions under which stockholders and security holders who do not appear upon the books of the company as tructees, hold stock and securities in a capacity other than that of a bona fide owner; and this affiant has no reason to believe that any other person, association, or corporation has any interest direct or indirect in the said stock, bonds, or other securities than as so stated by him.

C. F. KARSTAEDT, Secretary-Treasurer.

Sworn to and subscribed before me this 4th day of October, 1938.
(SEAL)

GRACE E. TROON.
(My commission expires February 22, 1942.)

them for the further handling of the case. The medical witness frequently makes or breaks the case by being too partial in his statement of theories and not the facts in the case, and also allowing the jury to think he is too one-sided in his views if he is employed by a railroad company.

Develop a reputation for being frank, fair, impartial in your remarks, thoroughly versed on the subject you are being examined on, and you will be successful.

Never be afraid to answer "I do not know."

Assistance To Railroad and Public

A RAILROAD surgeon of the type I have described occupies an enviable position in the personnel of the railroad company, and is a benefactor to any city or community that is fortunate enough to claim him as a citizen. A surgeon of the type described can be of valuable assistance to the railroad company in many ways.

In any city or community of 5,000 or less inhabitants with the exception possibly of the agent, the railroad surgeon should be of more value to the railroad company than any of the railroad personnel located there.

His education and follow-up study makes him above the average of his fellow men. His professional ability, his knowledge of human nature and his knowledge of personal, financial and political affairs of people in his city, make him of exceedingly valuable assistance to the railroad company.

His reputation as a man and a physician, his knowledge of political and financial conditions of the people of his city, coupled with the broadminded sympathies and understanding viewpoints from which the physician analyzes conditions, make him an outstanding man, and a man in a position to render assistance to both the railroad company and the people in his city. Regarding the assistance that this type of man can give to the community, I call your attention to the following: He serves the rich and poor alike. He is, as a rule, an outstanding man in civic, fraternal, financial, and political affairs. Primarily, he has a good education, and this advantage is enhanced by keeping abreast of the time in his own profession, and, due to the close intertwining of his own profession with the affairs of the world, he keeps pace with the affairs of the world as a whole. The teaching of a physician, and the daily application of these teachings in his professional work, make him analyze situations, not only from a professional standpoint, but also from a general standpoint, and thereby gives his community the benefit of a trained analytical mind to help solve its problems.